VOLUME 42 NUMBER 1

ARCHIVES OF

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Mirror-coated lenses have proven very successful in giving the high myope glare protection without the unsightly appearance which usually is associated with minus sun glasses. The coating hides the fact that the centers of such lenses are almost white while the edges are very dark.

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A yachtsman recently complained that the glare from the water was very annoying especially in early morning and late afternoon. As an experiment we tried coating the bottom one-third of his lenses as well as the top third. While he has not had a lot of time to try them out, the few times he did use them he was very well satisfied.

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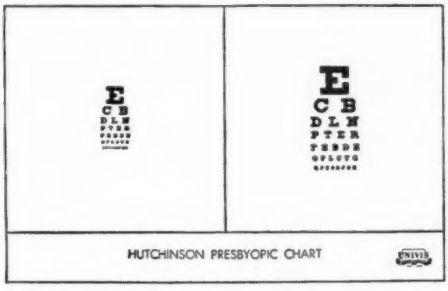

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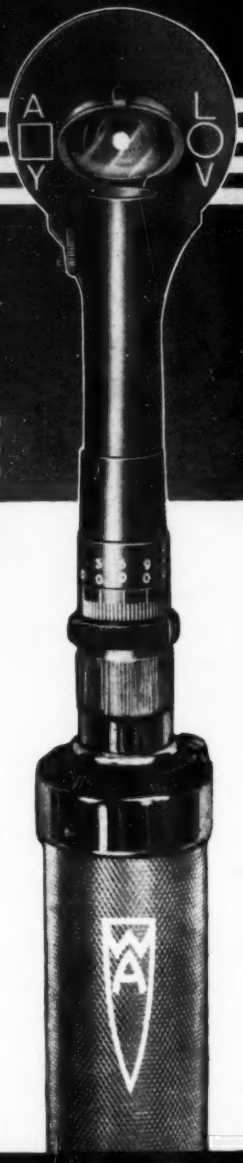

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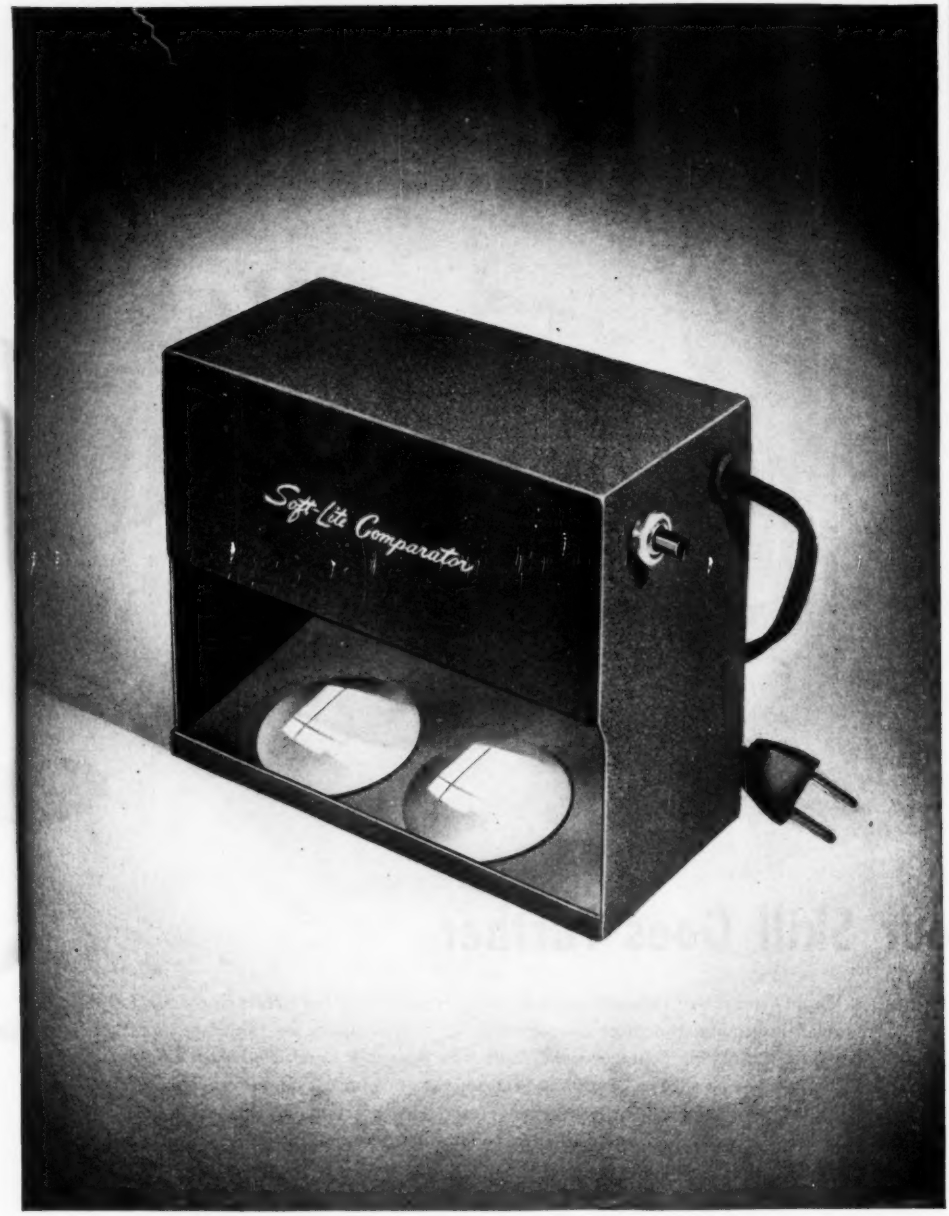

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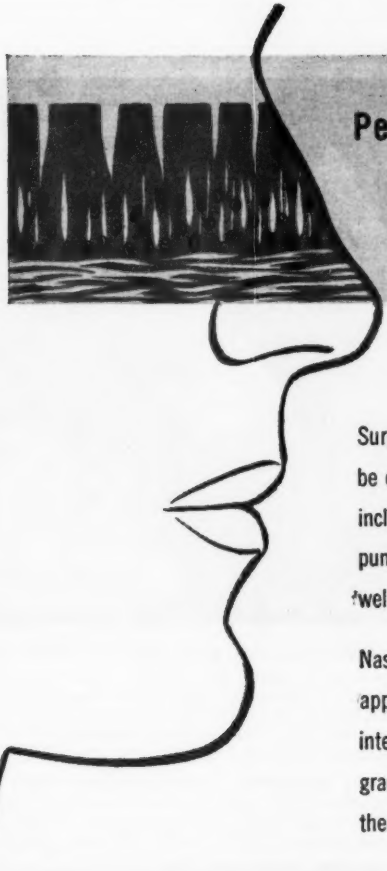

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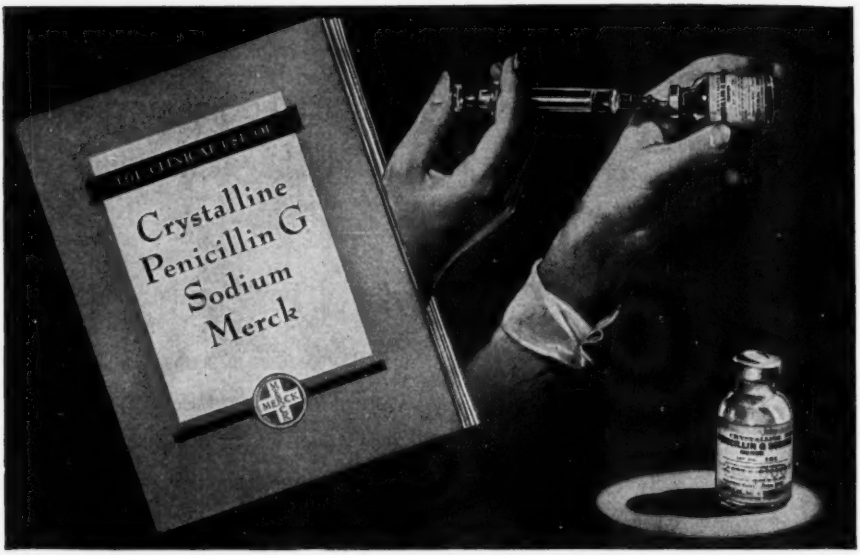

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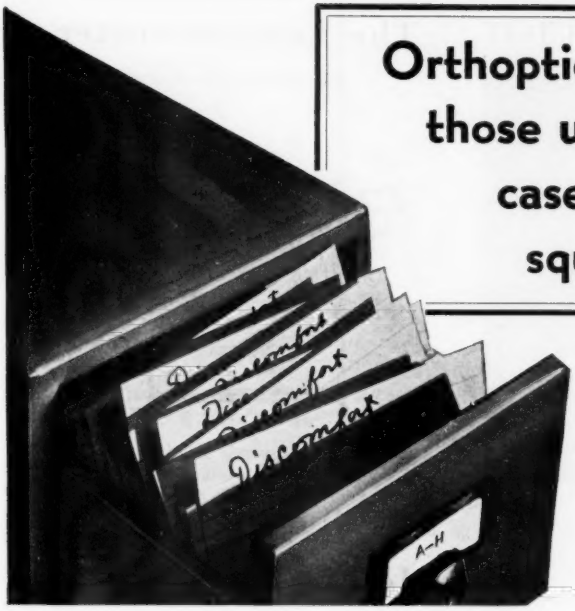

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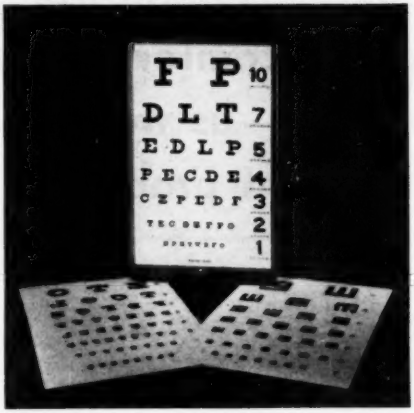

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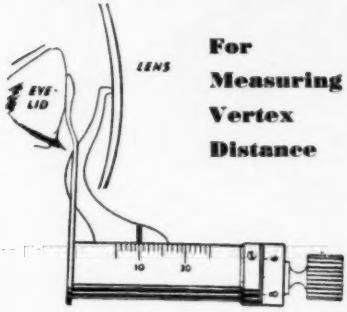

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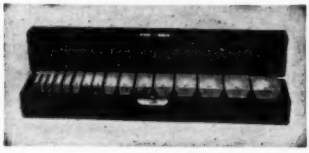

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ARCHIVES OF OPHTHALMOLOGY

VOLUME 42 JULY 1949 NUMBER 1

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INTRAORBITAL ANEURYSM

A Case of Aneurysm of the Lacrimal Artery

ROBERT F. HEIMBURGER, M.D.

H. R. OBERHILL, M.D.

H. ISABELLE McGARRY, M.D.

AND

PAUL C. BUCY, M.D.

CHICAGO

SURVEY OF LITERATURE

ANEURYSM within the orbit is a rare condition, in spite of the fact that it is commonly referred to in any discussion of the differential diagnosis of exophthalmos and that a number of cases, so diagnosed, have been reported in the literature. In a survey of the literature we have found reports of 68 cases in which a diagnosis of intraorbital aneurysm was made. Very few of these reports contain information concerning any pathologic investigation, and in even fewer do the recorded facts indicate that a true intraorbital aneurysm was present. In the great majority of instances the diagnosis rested on clinical observations alone.

In the early ophthalmologic literature it is evident that the diagnosis of intraorbital aneurysm was usually based (of course, erroneously) on the finding of a pulsating exophthalmos. In 1823 Guthrie [1] made the first diagnosis of orbital aneurysm, basing his diagnosis on autopsy observations. His was the third recorded case of pulsating exophthalmos, being preceded by cases of Travers [2] and Dalrymple,[3] in both of which the diagnosis was "aneurysm by anastomosis." In 1839 Busk [4]

From the Chicago Memorial Hospital and the Department of Neurology and Neurological Surgery of the College of Medicine of the University of Illinois.

1. Guthrie, G. J.: Aneurysms Within the Orbit: Lectures on the Operative Surgery of the Eye, London, S. Gosnell, 1823, pp. 157-158.

2. Travers, B.: A Case of Aneurysm by Anastomosis in the Orbit, Cured by Ligation of the Common Carotid Artery, Med.-Chir. Tr., London **2**:1, 1809.

3. Dalrymple, W.: A Case of Aneurysm by Anastomosis in the Left Orbit, Med.-Chir. Tr., London **6**:111, 1815.

4. Busk, G.: A Case of Aneurysmal Tumor in the Orbit, Med.-Chir. Tr., London **22**:124, 1839.

reported 2 cases of pulsating exophthalmos and, without supporting pathologic evidence, coined the phrase "true aneurysm in the orbit" as the diagnosis. The greatest impetus was given to this diagnosis by Sir Thomas Nunneley,[5] who in 1859 reported 4 cases of pulsating exophthalmos. He expressed the opinion that "true aneurysm in the orbit" was the cause of the condition in all 4 cases, even after his only autopsy revealed an aneurysm of the internal carotid artery within the carotid sinus. In 1865 Nunneley [5b] described 2 other cases of pulsating exophthalmos, caused in one by an aneurysm of the internal carotid artery and in the other by intracranial extension of a carcinoma of the thyroid gland. In the light of the autopsies in these 2 cases and in the one which he had reported previously, Nunneley revised his earlier opinion and concluded that pulsating exophthalmos is caused by any mass which compresses the ophthalmic vein as it emerges from the orbit. Although in this view he was still in error, he at least had begun to realize that the pathologic process in cases of pulsating exophthalmos usually lies outside the orbit. However, this change in Nunneley's opinion had little effect on the English and American authors, and reports, without pathologic proof, of aneurysm within the orbit continued to appear.

During the same period Baron [6] led a group of French surgeons in gathering data to show that arteriovenous fistula is the commonest cause of pulsating exophthalmos. In 1835 he demonstrated for the first time at autopsy a communication between the internal carotid artery and the cavernous sinus, suggesting the rupture of an aneurysm of the internal carotid artery as the basis of the lesion. His brief report went largely unnoticed, as did that of Henry [7] (Nélaton's case), who in 1855 also found a communication between the internal carotid artery and the cavernous sinus at autopsy in a case in which pulsating exophthalmos developed after the orbit had been pierced by the rib of an umbrella. It required a review by Delens [8] of all the cases of pulsating exophthalmos reported before 1870 to turn the tide of thought toward intracranial arteriovenous fistula as the common cause of pulsat-

5. Nunneley, T.: (*a*) Aneurysm of or Within the Orbit: Account of Three Cases, Med.-Chir. Tr., London **42**:165, 1859; (*b*) Vascular Protrusion of the Eyeball, ibid. **48**:15, 1865.

6. Baron: A Case of Ruptured Internal Carotid Aneurysm in the Cavernous Sinus, Bull. Soc. anat. de Paris **10**:178, 1835.

7. Henry (interne de l'hôpital): Note sur un aneurysme arterioveneux (communication de carotid interne avec le sinus caverneux) observation dans la service de M. Nélaton, Bull. Soc. anat. de Paris **30**:178, 1855.

8. Delens, E.: De la communication de la carotid interne et du sinus caverneux (aneurysme arterio-veneux), Paris, Adrien Delahaye, 1870.

ing exophthalmos. Delens' monograph was followed by similar collections of cases by Rivington[9] (1875), Sattler[10] (1880), Dempsey[11] (1886), de Schweinitz and Holloway[12] (1908) and Rhodes[13] (1916), each case strengthening the diagnosis of arteriovenous fistula. Since then, reports of intraorbital aneurysm have been less and less frequent, until, except for a brief reference by Rand[14] in 1944, the last one, by Vail and Oliver,[15] appeared in 1914. Even in Vail and Oliver's case the diagnosis was presumptive and was not based on actual demonstration of the aneurysm; and in Rand's case available information was inadequate to establish the diagnosis.

In a report by Locke[16] in 1924, who collected 588 cases of pulsating exophthalmos, arteriovenous fistula within the cranial cavity was discussed almost to the exclusion of aneurysm within the orbit. Intraorbital aneurysm was mentioned only briefly, in the historical note. The same is true of the report published by Martin and Mabon[17] in 1943, on 812 cases of pulsating exophthalmos. Since these two reports, the term "pulsating exophthalmos" has become almost synonymous with a diagnosis of fistula between the internal carotid artery and the cavernous sinus. That this trend is almost as erroneous as the old diagnosis of aneurysm within the orbit is shown by Locke's analysis of the 50 autopsies performed in 588 cases which he collected. In 32, or 64 per cent, of these autopsies the pulsating exophthalmos was found to be due to an intracranial arteriovenous fistula, while the remaining 18, or 36 per cent, revealed that intraorbital tumor or simple aneurysm, just posterior to the orbit, caused the condition.

We have found in the literature reports of 6 cases in which the diagnosis of intraorbital aneurysm was based on pathologic changes seen at autopsy or at operation. Few of these reports give satisfactory evi-

9. Rivington, W.: Pulsating Tumor of the Left Orbit, Med.-Chir. Tr., London **58**:183, 1875. (Comments are appended with all recorded cases of intraorbital aneurysms.)

10. Sattler, H.: Pulsirender Exophthalmus, in Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1880, vol. 6, p. 745.

11. Dempsey, A.: Case of Orbital Aneurysm, Brit. M. J. **2**:541, 1886.

12. de Schweinitz, G. E., and Holloway, T. B.: Pulsating Exophthalmos, Philadelphia, W. B. Saunders & Company, 1908.

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15. Vail, D., and Oliver, J. C.: Aneurysm of the Orbit in an Elderly Woman, Cured by Operation, Lancet-Clin. **112**:644, 1914.

16. Locke, C. C., Jr.: Intracranial Arteriovenous Aneurysms, Pulsating Exophthalmos, Ann. Surg. **80**:1-24 and 272-280, 1924.

17. Martin, J. D., Jr., and Mabon, R. F.: Pulsating Exophthalmos, J. A. M. A. **121**:330 (Jan. 30) 1943.

dence that the cause of the exophthalmos was a simple aneurysm limited to the orbital cavity. To show the reasoning which brought us to this conclusion, the 6 cases will be discussed separately.

CASE 1.—Guthrie's [1] case, reported in 1823, is conceded by most authors to be a verified case of intraorbital aneurysm. The entire report of his case, quoted here, will explain the questions which have arisen regarding his diagnosis.

I have seen one case of true aneurism of the ophthalmic artery, of both sides, which terminated fatally. The symptoms were similar to those above mentioned, but no tumor could be perceived, the eye was gradually protruded until it seemed exterior to the orbit, but vision was scarcely affected. The hissing noise in the head could be distinctly heard, and was attributed to the aneurism. On death of the patient, an aneurism of the ophthalmic artery was discovered on each side, of about the size of a small nut; the vena ophthalmica cerebralis was greatly enlarged, and obstructed near where it passes through the foramen lacerum orbitalis superius, in the consequence of the great increase in the size the four recti muscles had attained, accompanied by almost cartilaginous hardness, which had been as much concerned in protrusion of the eye as the enlargement of the vessels. The disease existing on both sides prevented an operation on the carotid being attempted, to which indeed the patient would not have submitted.

Delens [8] was the first to question Guthrie's diagnosis, observing that the location of the arterial dilatation, in relation to the bony structures, was not definitely stated. He also expressed the opinion that a more complex lesion than a simple aneurysm was suggested by several statements in the report. Dempsey [11] pointed out that no examination of the cavernous sinus was mentioned. Rivington [9] was not satisfied with the clinical data given. Although the statement that there was an aneurysm on each ophthalmic artery "of about the size of a small nut" seems clear enough, there are other phases of this report which raise some doubts in one's mind: First, bilateral aneurysms of the same size on the same vessels constitute a rare, almost unheard of, coincidence. Second, simple aneurysm practically never produces "hissing" or any other type of noise. The occurrence of such a noise strongly suggests that the patient has an arteriovenous communication. Third, the increase in size of the extraocular muscles and their hardness suggest that the patient may have had a malignant exophthalmos of the type which is sometimes associated with thyroid disease (Naffziger). Lastly, the report is incomplete, and there is no information as to the state of the intracranial vessels, notably the internal carotid artery and the cavernous sinus.

CASE 2.—Carron du Villards [18] in his presentation to the Société anatomique de Paris in 1838, described the case of a woman whom he saw only after her death from a disease of the lower portion of the abdomen. Autopsy revealed an aneurysm of the ophthalmic artery just at the point where the vessel entered the orbit. The author did not state whether the aneurysm was intracranial or intraorbital but did say that it was the size of a small nut. He was unable to obtain any clinical data regarding the affected eye. Delens stated the belief that this case was similar to the one reported in 1861 by Demarquay,[19] in which an aneurysm of the intracranial portion of the internal carotid and ophthalmic

18. Carron du Villards: Guide pratique pour l'étude et le traitment des maladies des yeux, Paris, 1838, vol. 1, p. 484.

19. Demarquay: Traité des tumors de l'orbit, Schmidt's Jahrb. **112**:262, 1861.

arteries was observed at autopsy. Sattler [10] presented Carron du Villards' case as questionably one of intraorbital aneurysm.

Case 3.—The lesion in Passavant's [20] case (1866), thought by Sattler to be an intraorbital aneurysm, was diagnosed preoperatively as an aneurysm of the lacrimal artery. The patient, a 9 year old girl, was struck by a knitting needle thrown at her by any angry sister. The needle penetrated the lateral aspect of the lower eyelid. A short time later there developed 6 mm. of exophthalmos and pain in the wounded eye. The condition was thought to be due to an aneurysm of the lacrimal artery because the eye had lost all mobility and was turned strongly inward. Passavant operated, using a lateral approach, similar to the one described later by Krönlein.[21] No dilated vessels were apparent in the lateral portion of the orbit, but Passavant was able to feel a large dilated vessel medial to the optic nerve. He thought that this vessel was the ophthalmic artery but was unable to see or to ligate it. The patient's condition was not improved by his operation. His postoperative conclusion was that the exophthalmos was due to a complex vascular lesion, which he called *l'aneurism diffuse primatif*, a term used in connection with arteriovenous fistula. Certainly, there must be more than a little doubt as to the exact nature of the pathologic anatomy in this case.

Case 4.—Lewis [22] ligated the dilated ophthalmic artery in a case which he reported in 1907. The patient, a 26 year old man, had been unconscious for an unstated period after a blow on the back of the head. Several weeks after he again became conscious he discovered that his left eye was more prominent than his right. A sensation of "beating" extended throughout his head, increasing in intensity over a period of a year. When Lewis first saw him, there were 6 mm. of proptosis, diplopia, engorgement of the conjunctival and retinal vessels, a bruit and a palpable, pulsating tumor, which could be felt through the upper eyelid. At operation, Lewis used an orbital approach to the lesion. A quotation from his operative report follows:

"The [ophthalmic] artery was found to have curved upon itself, the largest portion having the diameter of a man's little finger. The dissection was long and difficult. The eyeball was everted as far as possible to the temporal side, where the aneurysmal tumor was found to extend backward, gradually reducing in diameter until it wound around the optic nerve near the optic foramen. Several ligatures were placed around the enlarged vessel as the operation proceeded, until at the last with some difficulty the artery was tied off just within the orbital cavity. . . . The interest in this case centers in the fact that, although some portion of the aneurysm extended outside the orbit, a ligature within succeeded in controlling it, and that without removal of the eye."

The situation here, too, is not clear. The facts that the condition developed after trauma, that there was a bruit, that the eye pulsated and that the conjunctival and retinal vessels were engorged all strongly suggested that the man actually had an arteriovenous communication and that the vessel which was gradually

20. Passavant: L'aneurism diffuse primatif, in de Wecker, L.: Traité théorique et pratique des maladies des yeux, Paris, Adrien Delahaye, 1866, vol. 1, pp. 733, footnote 2.

21. Krönlein, R. U.: Zur Pathologie und operativen Behandlung der Dermoid der Orbita, Beitr. z. klin. Chir., **4**:149, 1889.

22. Lewis, F. P.: Pulsating Exophthalmos, Ligation of the Orbital Artery: Recovery, Ophth. Rec. **16**:66, 1907.

occluded by repeated ligation was a dilated vein receiving arterial blood and pulsating because of the pathologic communication.

CASE 5.—Moutinho [23] reported in 1907 the successful treatment of pulsating exophthalmos by ligation of the common carotid artery. The exophthalmos appeared thirteen years after trauma to the eye had produced a cataract. In 1908 the patient died of pulmonary tuberculosis and multiple multiloculated cerebral abscesses underlying an old fracture of the temporal bone. Moutinho reported the autopsy observation under the title "verificação pela autopsia da cura d'un aneurisma da orbita,"[23b] The orbital veins were found to be greatly dilated, but there was no report on the arteries. Immediately beneath the anterior clinoid process on the side corresponding to the pulsating exophthalmos was an aneurysm of the internal carotid artery. It was the size of an almond, round, very hard and filled with an organized clot. Although it was within the cavernous sinus, no point of communication could be found between the artery and the vein. No mention was made of an aneurysm within the orbit. It is obvious that this case was one of a primary intracranial rather than of an intraorbital lesion.

CASE 6.—In Dempsey's [11] case there was undoubtedly an intraorbital vascular anomaly, but this was only part of an extensive malformation of the internal carotid artery. In addition to a sacculated aneurysm, the size of a "mandarin orange," within the orbit, there were an abscess of the middle cranial fossa and an aneurysm of the internal carotid artery extending from the entrance of the vessel into the cranial cavity to the origin of the ophthalmic artery. The ophthalmic artery was dilated to four times its usual size up to the point of its entrance into the orbit. The entire circle of Willis was dilated, as was the petrosal sinus on the opposite side. The ophthalmic artery on the opposite side was of normal size. No arteriovenous fistula could be found in this case.

Rand [14] briefly noted a case of aneurysm of the ophthalmic artery. The patient complained frequently of pain behind the left eye, accompanied with a noise in her head and a premonition of death. A few minutes after she had experienced excruciating pain, her left eyeball was observed to bulge, and she died shortly thereafter of a ruptured aneurysm. The report of the autopsy, with which Dr. Rand has supplied us, does not provide sufficient information to permit of a definite diagnosis.

Other cases have been referred to as instances of verified intraorbital aneurysm by authors collecting cases of pulsating exophthalmos. When the original articles reporting these cases are consulted, it is obvious that the pathologic findings do not support the diagnosis. Locke made the statement:

> When in 1839, Busk confirmed Guthrie's findings by autopsy of another case, it soon became accepted, at least in England, that aneurysms of the ophthalmic artery were the cause of this clinical complex.

This statement was repeated by Martin and Mabon,[17] using "Burke" instead of "Busk." In his paper, Busk reported 2 cases of pulsating

23. Moutinho, M.: (*a*) Aneurisma da orbita esquerda, Med. contemp. **25**: 90-93, 1907; (*b*) Verificação pela autopsia da cura d'un aneurisma da orbita, ibid. **26**:34-36, 1908.

exophthalmos, in both of which ligation of the carotid artery resulted in cure. In each case Busk made the diagnosis of intraorbital aneurysm, but neither patient died while under his care, so that no autopsy was performed. Sattler expressed the opinion that both Busk's patients had arteriovenous fistula; with this we would agree.

Lansdown [24] and Terry and Fred [25] each reported a case of fistula between the nasal artery and the ophthalmic vein. In both these cases a pulsating mass presented in the inner canthus of the eye and the lesion was cured by the local ligation of the feeding vessels. These cases probably represent arteriovenous fistula within the orbit.

It is obvious from this review of the literature that, although the diagnosis of intraorbital aneurysm has been made frequently, it has rarely been confirmed. In the vast majority of instances in which such a diagnosis was made as a causative factor in pulsating exophthalmos, a review of the case would point to an arteriovenous fistula as the most likely cause of the disturbance.

In the case to be presented an intraorbital aneurysm was found at operation and removed. The severe, nonpulsating exophthalmos, with paralysis of the extraocu'ar muscles, was completely relieved.

REPORT OF CASE

Nonpulsating exophthalmos of the left eye, of two months' duration.

Examination: Severe proptosis of the left eye; distention of the periorbital, conjunctival and retinal vessels; paralysis of ocular movements; blurring of optic disk; visual acuity, 20/20 (right eye) and 20/70 (left eye); ptosis of left upper eyelid; pupil small and sluggish.

Operation: Transcranial exploration of orbit and removal of aneurysm of lacrimal artery. Complete recovery.

A. M., a white woman aged 58, single, a housekeeper, gave a history of congestion of the left eye, which had appeared three weeks before, and of soreness of the eye, starting four or five days prior to her first office visit to one of us (H. M.), in December 1946. Edema of the bulbar conjunctiva with mild congestion was present in the lower half of the globe. Visual acuity was 20/40 in the affected eye. The nerve head and the vascular ratio were regarded as normal. The intraocular pressure was increased, measuring 37 mm. (Schiøtz). Five days later proptosis was noted. The conjunctival vessels were tortuous. At the same time the retinal veins were beginning to show distention. The visual field for the affected eye showed slight concentric constriction. Roentgenographic studies of the orbits and paranasal sinuses revealed a normal condition. On Jan. 17, 1947, proptosis was pronounced, and there was limitation in motion down and to the left and up and to the right. The patient was hospitalized and seen in consultation

24. Lansdown, F. P.: A Case of Varicose Aneurysm of the Left Orbit, Brit. M. J. **1**:736, 1875.

25. Terry, T. L., and Fred, G. B.: Abnormal Arteriovenous Communication in the Orbit, Involving the Angular Vein, Arch. Ophth. **19**:90 (Jan.) 1938.

with Dr. Peter C. Kronfeld, who stated that orbital venous thrombosis should be ruled out. After a week of treatment with heparin, there was no change in the condition of the eye, and it was concluded that the space-occupying lesion in the orbit was either an aneurysm or an hemangioma; and a transcranial exploration of the orbit was thought advisable. The intraocular pressure was consistently elevated; the veins in the retina had become greatly distended, and large, flame-shaped hemorrhages had developed in all four quadrants of the fundus. The exophthalmometric reading (Luedde) measured 10 mm. in the right eye and 18 to 19 mm. in the left eye. The patient was admitted to the Chicago Memorial Hospital on February 27 for exploration.

The past history was noncontributory, except for the statement that the patient had been treated for bilateral tinnitus for about one year before the onset of the exophthalmos. The tinnitus had diminished with treatment but had not disappeared.

The family history did not reveal any case of an orbital lesion.

Physical Examination.—On her admission to the Chicago Memorial Hospital (February 27), the patient was well developed and well nourished. She did not appear ill. There was a small palpable lymph node in the left side of the neck. The breasts, heart, lungs and abdomen were normal. The blood pressure was 120 systolic and 80 diastolic; the pulse rate was 68 per minute.

The left eye protruded. The periorbital skin was edematous and reddened. The conjunctiva was chemotic and intensely injected. There were pronounced ptosis of the left upper eyelid and severe limitation of the ocular movements laterally, medially and upward. The left pupil was smaller than the right and reacted sluggishly to light and in accommodation. There were slight blurring of the margins of the left optic disk and severe engorgement of the retinal vessels in the left eye. The peripheral visual fields were full, and the uncorrected visual acuity was 20/20 in the right eye and 20/70 in the left eye. The corneal reflex was present and normal bilaterally. In spite of the complaint of tinnitus, hearing was normal. The right eye was unaffected in any way.

Laboratory Examination.—The urine was normal. There were 4,450,000 red blood cells, 83 per cent hemoglobin and 7,250 white blood cells, with 66 per cent neutrophils. The Wassermann and Kahn tests on the blood gave negative reactions.

Roentgenologic Examination.—There were no demonstrable lesions in the orbits. Studies of the chest and the paranasal sinuses showed nothing abnormal.

Preoperative Impression.—The proptosis was thought to be due to an intraorbital mass, probably vascular.

Operation (February 28).—A coronal incision was made just posterior to the hair line, and a frontal osteoplastic flap was reflected on the left side. The dura mater was firmly adherent to the inner table of the vault of the skull, owing to the presence of hyperostosis interna frontalis. The frontal lobe was elevated extradurally to expose the roof of the orbit. The roof of the left orbit was removed, anteriorly to the vertical portion of the frontal bone, laterally to the lateral margin of the orbit medially to the ethmoid cells and posteriorly to the sphenoid ridge and the optic foramen. On palpation through the orbital capsule, a firm mass running anteroposteriorly behind the bulb in the lateral portion of the orbit could be felt. When the orbital capsule was opened, the fat protruded under considerable tension. The levator palpebrae superioris and

superior rectus muscles were dissected from the fat and retracted medially. Lying lateral and slightly inferior to these muscles, a grayish blue cylindric mass was discovered (fig. 1). This mass was about the diameter of a lead pencil

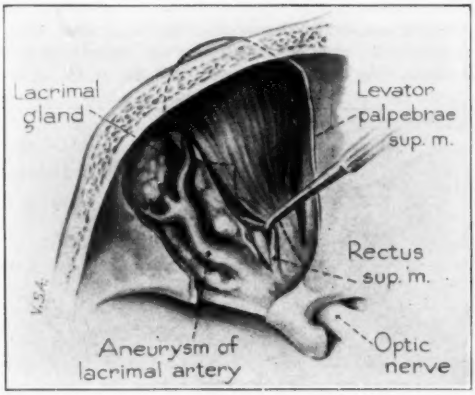

Fig. 1.—Artist's elaboration of the surgeon's sketch made immediately after the operation. The roof of the orbit has been removed. The aneurysm of the lacrimal artery is seen lying in the posterolateral part of the orbit.

Fig. 2.—The aneurysm in cross section. Hematoxylin-eosin stain; × 16.

and was 2 cm. in length. It pulsated synchronously with the heart beat. Anteriorly, it divided into three branches. The largest of these curved medially, and its

destination could not be accurately determined. The two lateral branches were smaller and entered the lacrimal gland. All three branches were divided between silver clips, and the anterior portion of the aneurysm was lifted from its bed. The posterior end of the lesion was in the region of the superior orbital fissure, the roof of which was removed to determine the relation of the vessels. The posterior end of the aneurysm was sectioned between a series of silver clips. The dura mater over the floor of the anterior cranial fossa was incised, exposing the orbital surface of the frontal lobe, the sylvian vessels and the tip of the temporal lobe.

Fig. 3.—The aneurysmal wall. The circular muscle fibers of the media are widely separated by edematous tissue. The longitudinal bundles of muscle fibers at the periphery are more compact. There is no layer of elastic tissue. Hematoxylin-eosin stain; × 80.

Medially, both anterior clinoid processes and the optic nerves were clearly seen. The intracranial vessels were of normal size and relation. No vascular dilatation or other abnormality was seen. The orbital capsule was left open; the frontal lobe was returned to the anterior cranial fossa, and the bone flap was replaced and secured with sutures.

Pathologic Report.—The specimen consisted of a blood vessel 2 cm. in length and 4 mm. in diameter (fig. 2). Microscopically, the aneurysm had a distinctly

abnormal wall. The media was very much thickened and its muscle was myxedematous (fig. 3). The elastic layer was not present.

Postoperative Diagnosis.—The diagnosis was aneurysm of the left lacrimal artery, contained entirely within the orbit.

Postoperative Course.—Mental aberrations appeared on the fifth postoperative day. These "frontal lobe signs" persisted for three or four days and then subsided completely. At the patient's discharge on March 13, her speech and memory were normal. There was postoperative increase in periorbital edema, but this was subsiding rapidly when she left the hospital. There was moderate ptosis of the left upper eyelid at that time.

On March 27, the patient was feeling very well. At that time, the left pupil reacted sluggishly; there was pulsation of the retinal arteries in the left eye synchronous with the pulse, and many superficial hemorrhages appeared along the course of the veins in all quadrants. The intraocular pressure measured 26 mm. (Schiøtz), being normal for the first time since the proptosis had been noted, prior to operation. The exophthalmometric reading was 10 mm. in the right eye and 13 mm. in the left eye.

On April 30, corrected visual acuity was 20/20 in the left eye. There was slight exophthalmos. Ocular motility was normal, as was the intraocular pressure. The retinal hemorrhages were no longer visible.

On July 8, the patient still noticed slight exophthalmos, but only when she became tired. There was no complaint regarding vision, and the ocular movements were unimpaired.

In December 1947, the peripheral fields on the left still showed 5 to 15 degrees of concentric constriction. The intraocular pressure and the ratio of the retinal vessels were normal.

On Jan. 7, 1948, there was no complaint referable to the left eye. The patient commented, however, that the eye pulsated occasionally, especially when she became tired. The tinnitus had disappeared. There was slight enophthalmos. The ocular movements were full. The retinal vessels and the optic disks were normal. The uncorrected visual acuity was 20/40 in the left eye and 20/30 in the right eye. The peripheral fields still showed concentric constriction on the left. Roentgenographic examination of the orbit revealed the silver clips which occluded the arterial communications of the aneurysm, and the defect in the bone, but no other abnormality.

COMMENT

A tabulation of signs and symptoms which would lead to a differentiation of intraorbital aneurysm and intracranial arteriovenous fistula cannot be made from the small number of cases, many of them questionable, of intraorbital aneurysm which have been reported up to the present time. Intraorbital aneurysm is certainly a rare condition. The reasons for this rarity pointed out by Nunneley [5b] in 1865 are still plausible: Arteries anywhere in the body of the caliber present in the orbit infrequently form aneurysms; and, in order to produce clinical manifestations, an aneurysm of the orbit would of necessity be relatively large, so as sufficiently to compress the areolar tissue which fills the

orbital cavity. Nunneley was aware that a small mass just posterior to the orbit could produce exophthalmos by compromising the venous blood flow, but he made no mention of similar conditions occurring just within the orbit.

The origin of the aneurysm in the case reported here is not clear, but the lack of elastic tissue in the arterial wall suggests a condition similar to that in the congenital aneurysms of the intracranial vessels. The development of the first symptoms during the sixth decade of life is unusual in the case of a congenital lesion, but it is not unheard of with intracranial aneurysms. The lacrimal artery most frequently arises from the ophthalmic artery soon after it enters the orbital cavity. It may, however, arise from the intracranial portion of the ophthalmic artery, from the middle meningeal artery or, more rarely, by a large anastomosis between the lacrimal branch of the middle meningeal artery and a recurrent branch of the ophthalmic artery. In these instances the lacrimal artery enters the orbit through the superior orbital fissure.[26]

The chemosis and injection of the conjunctiva, the distention of the retinal veins and the retinal hemorrhages are all evidence of venous obstruction. This interference with the venous drainage of the orbit was probably directly responsible for the exophthalmos, the impaired vision and the increased intraocular tension. Whether it resulted from mere compression of the veins, or from a venous thrombosis, as Dr. Kronfeld originally postulated, can now be only a matter for speculation.

As demonstrated by the experience of Passavant and Lewis, the orbital approach to vascular lesions of the orbit is dangerous and liable to failure. With the transcranial route advocated by Naffziger[27] and Dandy,[28] the entire lesion, both intracranial and intraorbital, can be visualized, with little danger to the eye or to the optic nerve.

As this review of the literature has clearly shown, exophthalmos is so frequently associated with intracranial disease, rather than with a lesion limited to the orbit, as to make the ability to explore the intracranial cavity a valuable addition to the operative procedure. Furthermore, the extent of the exposure by the transcranial route, the much better visualization of both intraorbital and intracranial structures and the greater ease and safety in controlling the blood supply make this the preferable surgical approach in many cases.

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26. Jackson, C. M.: Morris' Human Anatomy, ed. 9, Philadelphia, P. Blakiston's Son & Co., 1933.

27. Naffziger, H. C., and Jones, O. W.: The Surgical Treatment of Exophthalmos Following Thyroidectomy, J. A. M. A. **99**:638 (Aug. 20) 1932.

28. Dandy, W. E.: Orbital Tumors, New York, Oskar Piest, 1941; Results Following the Transcranial Operative Attack on Orbital Tumors, Arch. Ophth. **25**:191 (Feb.) 1941.

SUMMARY

A case of exophthalmos due to aneurysm of the lacrimal artery contained entirely within the orbit is reported. The lesion was removed by the transcranial route, and the patient is entirely well, over a year after operation, with normal vision and normal ocular movements in the affected eye.

A survey of the literature on orbital aneurysm revealed no other verified case of aneurysm of the lacrimal artery. Reports of 6 cases in which an intraorbital aneurysm was said to have been visualized either at operation or at autopsy are reviewed, but in none is the diagnosis without question. Because of this lack of verified cases, no criteria can be set up on which a differential diagnosis of an aneurysm within the orbit may be based. The case presented here is evidence that orbital aneurysm can be manifest clinically by unilateral exophthalmos without pulsation, but with evidence of venous obstruction, increased intraocular tension and failing vision.

25 East Washington Street.

VISUAL ACUITY WHILE ONE IS VIEWING A MOVING OBJECT

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FOVEAL visual acuity while one is viewing a moving object has received curiously little attention. In fact, the only reference to this subject which I have been able to find is a note by Langmuir [1] in which, in an effort to estimate the speed of a deer botfly, he whirled above his head a piece of solder fastened to a string and noted at what speed the piece of solder appeared blurred. I have discussed this problem.[2]

There have been investigations of the minimum velocity perceptible as movement [3] and the way in which this minimum velocity varies from center to periphery of the visual field.[4] There have also been investigations of the minimum length of path which permits motion to be perceptible.[5] Some have claimed [6] that there is "visual anesthesia" while the eyes are voluntarily moving. Bethe's "Handbuch," [7] Duke-Elder,[8] Troland [9] and Vernon [10] make no mention of any determination of visual acuity while the eye is following a moving object.

This investigation was supported in part by a grant from the American Optical Company.

From the Howe Laboratory of Ophthalmology and the Massachusetts Eye and Ear Infirmary.

1. Langmuir, I.: The Speed of the Deer Fly, Science **87**:233 (March) 1938.

2. Ludvigh, E. J.: Visibility of the Deer Fly in Flight, Science **105**:176 (Feb.) 1947.

3. Munck, G. W.: Gesicht und Sehen, in Physik. Wörterbuch **4**:1457, 1828.

4. Bourdon, B.: La perception visuelle de l'espace, Paris, Lille, 1902, p. 201.

5. Basler, A.: Ueber das Sehen von Bewegungen, Arch. f. d. ges. Physiol. **115**:582-601, 1906.

6. Holt, E. B.: Eye-Movement and Central Anaesthesia, Psychol. Rev. Monog. Supp. **4**:3-45, 1903. Dodge, R.: Visual Perception During Eye-Movement, Psychol. Rev. **7**:454-455, 1900.

7. Bethe, A.; von Bergmann, G.; Embden, G., and Ellinger, A.: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1931, vol. 12, p. 2.

8. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1932, vol. 1.

9. Troland, L. T.: The Principles of Psychophysiology, New York, D. Van Nostrand Company, Inc., 1929, vol. 1.

10. Vernon, M. D.: Visual Perception, London, Cambridge University Press, 1937.

In view of the absence of any information on this subject, it was thought advisable to conduct an investigation of the problem, particularly since the eyes are so often and critically employed under these conditions.

PRESENT INVESTIGATION

The simplest type of motion first to be investigated seemed to be simple rotary motion of the test object. This was produced by inserting prisms of various strengths in a ball bearing and then driving the ball bearing by means of a variable speed drive. The result was that the object viewed appeared to travel about in a circle in a plane perpendicular to the line of sight. Monocular observation was used. The test objects employed were Snellen letters. Eight letters were selected from various charts so that the same eight letters could be used on the 20/100, 20/70, 20/50, 20/40, 20/30, 20/20, 20/15 and 20/10 lines. The maximum distance of observation conveniently available was 5 meters. When the prism was rotated rapidly enough to decrease vision below 20/100, the 3 observers were moved up to a distance of 2 meters from the test object.

TABLE 1.—*Relation of Visual Acuity to Increasing Angular Velocity of the Test Object*

Angular Velocity of Letter Presented (Degree/Sec.)	Visual Acuity Expressed as a Fraction of the Acuity at 0°/Sec. Angular Velocity	
	4△	12△
0	1.000	1.000
12	0.887	0.886
25	0.538	0.806
38	0.204	0.675
50.4	0.103	0.621
61.5	0.083	0.456
75.6	.....	0.299

The experimental procedure was as follows: With the prism rotating at a given speed, the lowest line was determined which resulted in ten successive correct readings of the Snellen letters. The Snellen letters were presented in an order previously determined by the chance selection of cards. On the next lowest line, say the 20/20 line, suppose that seven of the ten letters were correctly read, one would reason, somewhat arbitrarily, that when three errors were made, had the letters been of 20/30 size, they would have been correctly read, since the subject had just previously read ten letters correctly on this 20/30 line. We concluded that on the ten occasions just tested, the subject exhibited 20/20 vision on seven occasions and 20/30 vision on three occasions and that the acuity was therefore intermediate between these two values; more specifically, that it was 20/22.2.

When the apparent motion of the letter is in a circle in a plane perpendicular to the line of sight, it is not immediately apparent what one should characterize the angular velocity of the letter. However, the simplest way of thinking about the matter would seem to be this: Suppose that the apparent movement of the letter is such that it travels around the circle, say five times per minute. Furthermore, suppose that the circumference of this circle is 10 cm. and that the distance of observation is 5 meters; then the letter is traveling 50 cm. per minute. If it were traveling not in a circle in a plane perpendicular to the line of view but, rather,

in a circle around the subject's head at a distance of 5 meters and at a speed of 50 cm. per minute, then it would be going around the subject's head at 5.73 degrees per minute; and it is in this fashion that the angular velocity of the object is defined when the rotating prism is employed.

Figure 1 shows the manner in which visual acuity deteriorates with increasing angular velocity of the test object, and the data are presented in table 1.

It will be observed that when a prism of 4 Δ is being rotated at a speed sufficient to cause an angular velocity of 62 degrees per second

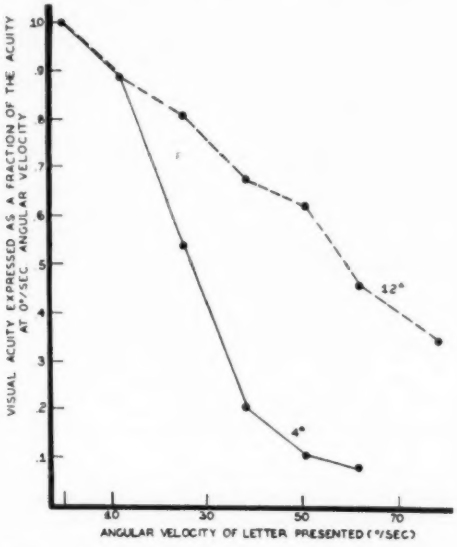

Fig. 1.—Deterioration of visual acuity with increasing angular velocity of the test object.

the visual acuity has been reduced from 20/20 to below 20/200 degrees. Also, in the figure are shown the results obtained with a 12 Δ prism. It will be observed that when an angular velocity of 62 degrees per second is produced by the use of a 12 Δ prism, vision is reduced to about 20/40. This means that when the large prism is employed, the same angular velocity of the letter produces much less deterioration of visual acuity than when the small prism is used. If one may greatly oversimplify the situation, in making a full rotation, innervation of the muscles occurs in the following order: first, the superior rectus muscle, then some of the internal rectus, then the internal rectus alone, then

some of the inferior rectus, and so on, with accompanying inhibitions of the antagonistic muscles. It may be, then, that the falling off of acuity while the eye is following an object moving in a circular path is attributable to inability to repeat the necessary cycle of innervation more than so many times per minute, and it appears, indeed, that this lack of ability to change innervation sufficiently rapidly is a cause of the deteriorating visual acuity with increased speed of motion of the test object. But that this is not the sole factor is shown by the experimental

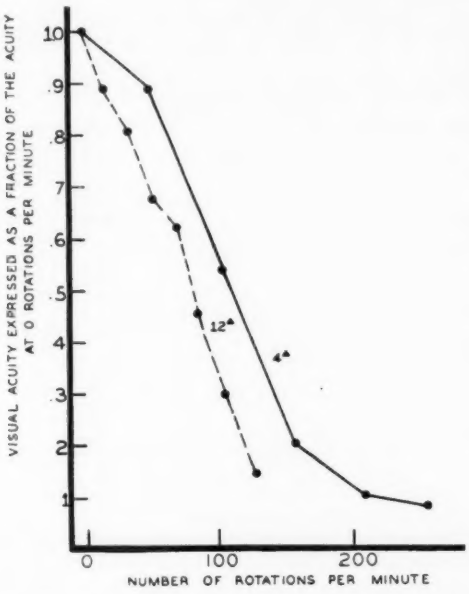

Fig. 2.—Relation of visual acuity to number of rotations per minute of the test object.

data presented in table 2 and shown graphically in figure 2. Here plotted on the horizontal axis is not the angular velocity of the letter in degrees per second, but the number of rotations of the letter per minute. Consider the point at which the number of rotations per minute is slightly in excess of 100. With the 4 Δ prism, the acuity is better than 20/40, while with the 12 Δ prism the acuity is 0.3, or about 20/70; yet the eye need make no greater number of rotations for the 12 Δ prism than for the 4 Δ prism, so that the percentage rate of change of innervation cannot here be the controlling factor. However, with the

12 Δ prism, the eye must travel through a considerably greater distance, and therefore at a higher angular speed. This indicates that the higher the angular velocity of the test letter, the more the acuity will be reduced. We conclude that when the eye follows an object moving about in a circle in a plane perpendicular to the line of sight, the acuity will be reduced by reason either of the inability of the eye to move sufficiently rapidly or of the inability of the subject to adjust the relative innervation to the various ocular muscles sufficiently rapidly or of both.

The question arises: What is the cause of the reduction of acuity when the eye is following a moving object? Perhaps the first hypothesis to be considered is that acuity decreases when the object is moving

TABLE 2.—*Relation of Visual Acuity to Number of Rotations per Minute of the Test Object*

Visual Acuity	Number of Rotations per Minute	Visual Acuity Expressed as a Fraction of the Acuity at 0 Rotations per Minute
4Δ	0	1.000
	50	0.887
	104	0.538
	158	0.204
	210	0.103
	256	0.083
12Δ	0	1.000
	16.6	0.886
	34.6	0.806
	52.6	0.675
	70.0	0.621
	85.3	0.456
	105	0.299
	123	0.147

because imperfect pursuit movements of the eye result in extrafoveal portions of the retina being utilized. An examination of the data shows that this hypothesis can account for only a negligible fraction of the loss of acuity experimentally observed. Consider the instance in which the 4 Δ prism is being rotated. Suppose that pursuit movements of the eye were completely ineffective and that the eye merely fixated the center of the circle, which is the path in which the Snellen letter is moving. Then, under these conditions, the letter would be only 2 degrees from the fovea. Figure 3 shows some of the results of my investigation of extrafoveal visual acuity as measured with Snellen test letters,[11] and the data are given in table 3. It may be seen that at 2 degrees from the fovea visual acuity has deteriorated only to about 0.8, or 20/25.

11. Ludvigh, E. J.: Extra-Foveal Visual Acuity as Measured with Snellen Test-Letters, Am. J. Ophth. **24**:303, 1941.

This is clearly insufficient by a factor of 5 to account for the loss of acuity produced by rotating the 4 △ prism. Therefore, when the eye is following a moving object, the extrafoveal position of the image is a negligible factor in the observed reduction of acuity.

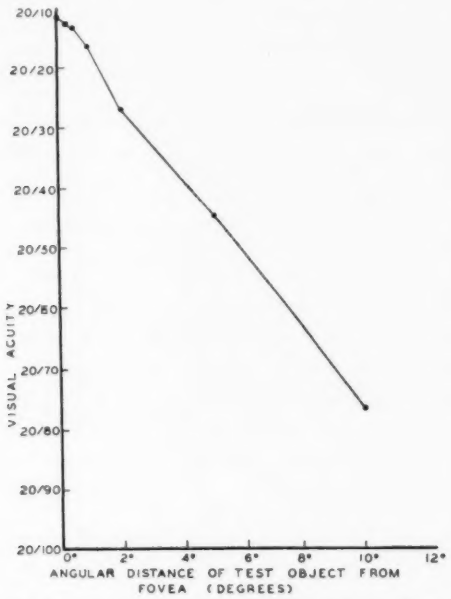

Fig. 3.—Relation of visual acuity to angular distance of the test object from the fovea.

TABLE 3.—*Relation of Extrafoveal Visual Acuity to Angular Distance of Test Object from Fovea*

Angular Distance of Test Object from Fovea (Degrees)	Visual Acuity
0	20/11.68
0.25	20/12.58
0.50	20/13.21
1.0	20/16.81
2.0	20/27.36
5.0	20/45.08
10.0	20/76.80

Another hypothesis is that imperfect pursuit movements of the eye, although maintaining the image in the immediate vicinity of the fovea, nevertheless result in a motion of the image on the retina which reduces

visual acuity. The eye is unable to match the exact rate of movement of the object, and hence the image is continually moving over the retina as the eye goes ahead of, and lags behind, the moving object.

If this hypothesis is correct, it is motion of the image on the retina which produces the deterioration of visual acuity. This motion would result in any given portion of the retina receiving less intensity contrast than it would if the eye and object were stationary. It might, therefore,

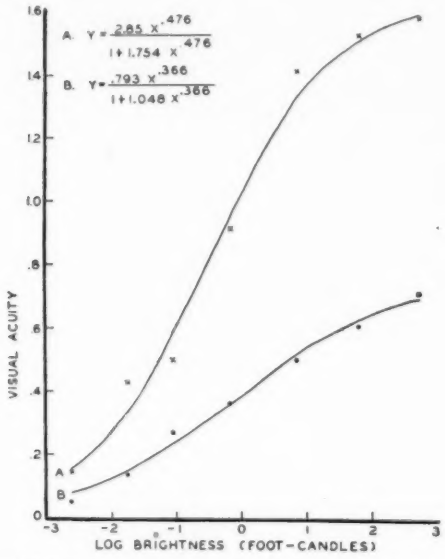

Fig. 4.—Relation of the visual acuity to intensity of illumination.

TABLE 4.—*Relation of Visual Acuity to Intensities of Illumination*

Log Brightness (Foot Candles)	Visual Acuity, Rotations/Min.	
	0	90
2.703	1.57	0.709
1.772	1.52	0.607
0.837	1.41	0.500
−0.197	0.916	0.360
−1.051	0.500	0.269
−1.764	0.428	0.132
−2.616	0.142	0.0427

be thought that high intensities of illumination would particularly aid visual acuity when the eye was moving. Figure 4 and the data in table 4 show that when the image is moving on the retina high intensities may

advantageously be employed. The test objects used in obtaining these data were Landolt rings. The brightly illuminated area subtended about 5 degrees at the eye, and for these conditions the characteristic sigmoid curve relating visual acuity and log intensity was found. It may be noted that the highest intensities of illumination produce little improvement in acuity when the object is stationary. This is indicated by the flattening out of the upper curve of the graph at high illuminations. The lower curve of the graph shows the results obtained when the test object is being displaced by a rotary prism at a velocity in path of 90 degrees per second. Here, even at the highest intensity of illumination available, about 505 foot candles, acuity is still improving. This suggests that, although for ordinary purposes about 10 foot candles is adequate illumination, an illumination of 1,000 foot candles might be advisable when the eye is following a rapidly moving object.

Since it is difficult visually to extrapolate a curve from the observed experimental points, the data were fitted by the method of least squares to the empiric function $y = \frac{ax^c}{1 + bx^c}$, where y is visual acuity and x is the illumination in foot candles on the chart. The use of the function $y = \frac{ax^c}{1 + bx^c}$ implies that visual acuity will become zero when the illumination is zero, and also that as the illumination becomes great visual acuity will approach $-\frac{a}{b}$ in value. No assumption is made as to nature of the process, photochemical or other, mediating the relation of brightness to visual acuity.

When the object is stationary, the data fitted by the method of least squares give rise to the curve $y = \frac{2.85\, x^{0.476}}{1 + 1.75\, x^{0.476}}$. The maximum value of visual acuity predicted by this formula is then $\frac{2.85}{1.75}$, or 1.62. Ninety per cent of this final value of visual acuity is 1.46, and this visual acuity is attained when the illumination is 30 foot candles. When the object is moving, the data fitted by the method of least squares give rise to the curve $y = \frac{0.793\, x^{0.300}}{1 + 1.05\, x^{0.300}}$. The maximum value of visual acuity predicted is 0.757, and 90 per cent of this value is not achieved until the intensity of illumination reaches 360 foot candles. It thus appears that a quantitative analysis of the data confirms the notion that high intensities of illumination might be advisable in following a moving object. This problem is being further investigated at still higher intensities of illumination.

SUMMARY

Visual acuity was determined while the test object viewed appeared to move in a circle in a plane perpendicular to the line of sight. The diameter of the circle and the velocity of the test object were varied. It is shown that when the eye is following such a moving object, acuity

is reduced by reason either of the inability of the eye to move sufficiently rapidly or of the inability of the subject to adjust the relative innervation to the various ocular muscles sufficiently rapidly or of both. It is shown that in following a moving object the extrafoveal position of the image is a negligible factor in producing the observed reduction of acuity.

The hypothesis is that imperfect pursuit movements, although maintaining the image in the immediate vicinity of the fovea, nevertheless result in a motion of the image on the retina which reduces visual acuity. This motion would result in the retina's receiving less intensity contrast than it would if the eye and object were stationary. The hypothesis tends to be confirmed by observations showing that when the test object is moving, high intensities of illumination may advantageously be employed to increase visual acuity.

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MECHANICS OF INTRACAPSULAR CATARACT EXTRACTION

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THERE can be little controversy among modern ophthalmic surgeons regarding the advantages and desirability of the intracapsular method of cataract extraction.

As Arnold Knapp [1] has pointed out in his classic paper:

> The amount of literature that has arisen on the subject of the intracapsular extraction is bewildering, but some general direction can be recognized. The trend seems to be in favor of an operation in which a firm hold is taken of the capsule with the forceps without tearing it, concentrating the traction to one area and with a hook or similar instrument exerting pressure externally at the lower corneal margin, in order to raise vitreous pressure and to rupture the suspensory ligament at that point. . . . After subluxation of the cataract, the extraction is completed with the aid of external pressure, by tumbling or by head-first delivery.

Knapp and numerous others have fully reviewed the advantages of intracapsular cataract extraction over the extracapsular method, with fewer and fewer dissenting voices in recent years. The "perfect cataract extraction," the goal of every ophthalmic surgeon, embraces all these advantages, which may be briefly outlined as follows:

1. A calm, cooperative patient on the operative table.

2. A more or less standardized operative technic, applicable to most types of cataract and fostering development of greater skill in both operator and assistant.

3. Routine employment of safety factors to assure a minimum of complications, both operative and postoperative.

4. A small, round, centrally placed, black pupil after operation, with minimum trauma to the iris.

Read at the Eighty-Fourth Annual Meeting of the American Ophthalmological Society, Hot Springs, Va., May 15, 1948.

From the Department of Surgery, Division of Ophthalmology, University of California Medical School, and the United States Veterans Administration Hospital.

1. Knapp, A.: Present State of Intracapsular Cataract Operation, Arch. Ophth. **38**:1 (July) 1947.

5. A tight wound, well protected against leakage, prolapse of the iris and hemorrhage and allowing rapid formation of the anterior chamber and early ambulation.

6. Absence of postoperative iridocyclitis and shortened period of convalescence.

7. Minimum astigmatism.

8. Maximum visual acuity in the highest percentage of cases.

9. A grateful patient, pleasantly surprised by the absence of an anticipated ordeal and anxious to undergo operation on the second eye in order to regain the benefits of binocular vision.

It is my belief, and there is ample support for it in the literature, that these criteria are satisfied only by the performance, on a psychologically prepared patient, of cataract extraction by the intracapsular method, through a round pupil, with the employment of all modern safety measures of proved merit. It is not the purpose of this paper to present a detailed operative technic or to claim superiority of one detail over another. The use of certain methods by way of illustration should be considered an advocacy not of those methods per se but only of the fundamental principles underlying them. This study is concerned only with the basic principles involved in the mechanics or dynamics of intracapsular cataract extraction and with the methods which have proved most valuable in utilizing certain physical principles to be demonstrated.

The classic physical experiment which demonstrates that in liquids a pressure at one point transmits itself to all points within the liquid with equal force is only partially true when applied to the vitreous gel in an eyeball opened for cataract extraction. The vitreous is a tissue, and the solider its consistency, the more readily can portions of it be "pinched off," so to speak, and made to act as isolated, smaller spheres without disturbance of the pressure ratio within the vitreous body as a whole. This fact can be demonstrated during the actual procedure of cataract extraction, on cadaver eyes, on animal eyes and by gonioscopic observation.

It is common knowledge among surgeons that the grasp of the intracapsular forceps on the anterior capsule of the lens is facilitated by pressure on the sclera sufficient to raise the vitreous pressure, thus pushing the lens forward into the jaws of the forceps. It can readily be demonstrated that the grasp on the lower portion of the lens capsule, at or under the inferior pupillary margin, preliminary to extraction of the lens by tumbling (fig. 1), is most easily obtained by applying pressure on the sclera well back toward the equator and directed upward and forward, as though "lifting" the vitreous. The lens will be seen to come forward, but it will also tilt or rotate on its horizontal axis, so that the inferior edge of the lens is anterior and against the jaws of the forceps

while the superior edge is tilted backward. A sufficiently large area of application of pressure is obtained by the Verhoeff ring [2] introduced deep into the lower fornix and applied flat to the globe.

On the other hand, if one wishes to apply the forceps to the upper edge of the lens, as is done in the Verhoeff [3] method of extraction, (fig. 2), pressure must be applied at the lower corneal limbus or even on the lower part of the cornea, and directed backward as though to push the vitreous back. This causes the lens to tilt or rotate on its horizontal axis, so that the superior edge is forward and may be straddled by the forceps while the inferior edge is depressed.

Further application of pressure in these maneuvers will cause the hydrostatic wedge of the vitreous to insert itself between the equator of

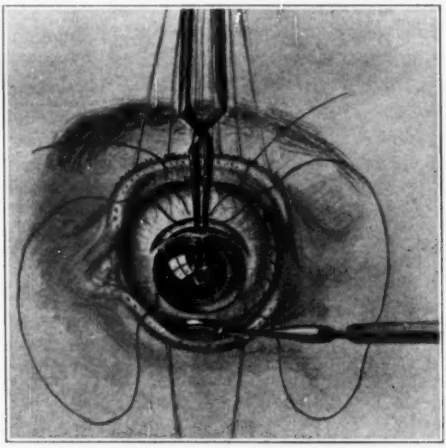

Fig. 1.—Low scleral pressure and applications of the capsule forceps in cataract extraction by tumbling.

the lens and the ciliary body and thus rupture the zonule (fig. 3 *A*), at the lower edge in the first instance and at the upper edge in the second. Light traction on the lens capsule, just sufficient to "tense" or stretch the zonule, will facilitate zonular rupture. Traction on the capsule alone without the aid of the wedge of vitreous will, of course, rupture the zonule in a certain percentage of cases, especially when it is abnormally weakened by age and the changes associated with cataract

2. Verhoeff, F. H.: Lens Expressor, Tr. Am. Ophth. Soc. **28**:305, 1930.

3. Verhoeff, F. H.: New Operation for Removing Cataracts with Their Capsules, Tr. Am. Ophth. Soc. **25**:54, 1927.

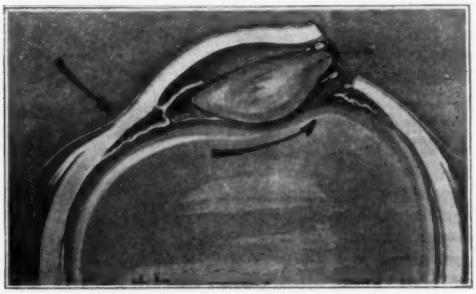

Fig. 2.—Direction of movement of the vitreous in the Verhoeff extraction with pressure on the corneal limbus.

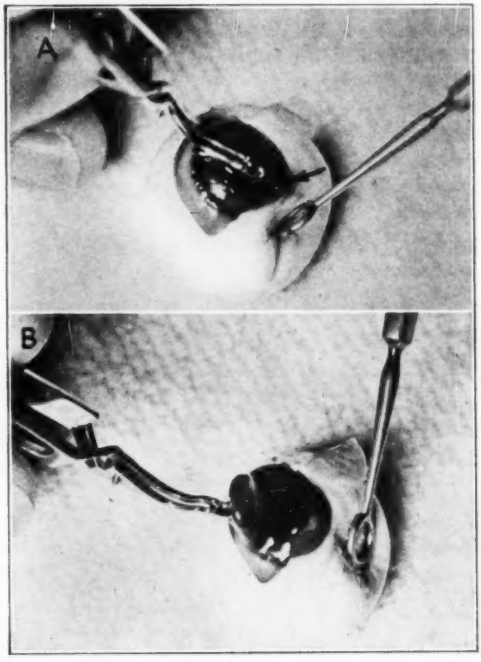

Fig. 3.—*A*, production of a wedge of vitreous between the inferior edge of the lens and the ciliary body, with rupture of the zonule. Pressure on the sclera was applied 8 mm. below the limbus. *B*, distortion of the vitreous with release of negative pressure in the hyaloid fossa as a result of low scleral pressure.

formation; but the incidence of capsular rupture is greater, the greater the ratio of traction to pressure, and one can almost state that "normally" the zonule would not be ruptured by traction alone.

After the rupture of the zonule has been accomplished, the lens must then be freed of its position in the hyaloid fossa, either by tumbling or by sliding it head first from the fossa. Barraquer [4] and Goldsmith [5] laid great emphasis on the strength of the negative pressure between the lens and the hyaloid membrane.

If the lens is tumbled, its disarticulation from the hyaloid fossa is accomplished by further application of pressure, "lifting" the vitreous below so that it presents into the lower part of the anterior chamber, pushing the lower edge of the lens ahead of it. At this point the distortion of the vitreous releases the negative pressure (fig. 3 *B*), and the lens continues to rotate on its horizontal axis. Now, for the first time, traction on the capsule sufficient to move the lens may be applied with little danger of capsular rupture, and the direction of pressure is shifted away from the vitreous and toward the cornea and the lens itself. The herniated vitreous immediately sinks back, and the final delivery of the lens is accomplished by a delicate balance between pressure and traction on the lens itself.

If the lens is to be delivered head first, as in the Verhoeff [3] operation, it must be freed from the hyaloid fossa primarily by traction. Application of pressure at the lower portion of the limbus sufficient to release the negative pressure in the fossa will present the vitreous ahead of the lens and into the wound (fig. 4). Pressure applied to the sclera will tend to elevate the inferior edge of the lens and depress the superior edge.

Goldsmith [5] objected strenuously to the head-first delivery of the lens by pressure and stated that only the fortunate previous subluxation of the lens superiorly, or early rupture of the superior zonular bundles, prevented disastrous prolapse and loss of vitreous. It should be pointed out that the Verhoeff procedure deliberately utilizes these "fortunate" factors by early zonular rupture with the forceps at the superior edge of the lens. During extraction, however, some pressure must be used, and the lens must be pressed against the posterior margin of the incision to hold the vitreous back and to avoid its loss.

The authority for the foregoing statements is the result of a gradual development of experimental work, which began several years before the war and has only recently been resumed.

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4. Barraquer, I.: Facoerisis, in Fisher, W. A.: Senile Cataract, Chicago, Chicago Eye, Ear, Nose, and Throat College, 1923, p. 34.

5. Goldsmith, J.: Dynamics of Intracapsular Cataract Extraction, Arch. Ophth. **29**:380 (March) 1943.

In 1930 I was afforded the opportunity of studying a patient with a very large iridodialysis opening, through which the equator of the lens and the zonular bundles were readily examined by slit lamp microscopy. Beyond the zonular barrier, which was apparently intact, the hyaloid membrane could be seen passing backward and laterally, with a vitreous of average normal consistency beneath it.

When pressure was applied to the corneal limbus, or 1 mm. on the cornea in the area of the iridodialysis, there was little or no movement in the zonular bundles or in the hyaloid membrane. When pressure was applied to the sclera at a point 6 to 8 mm. from the limbus on the same side as the area of iridodialysis, the visible vitreous seemed to move forward toward the site of iridodialysis, causing the hyaloid membrane

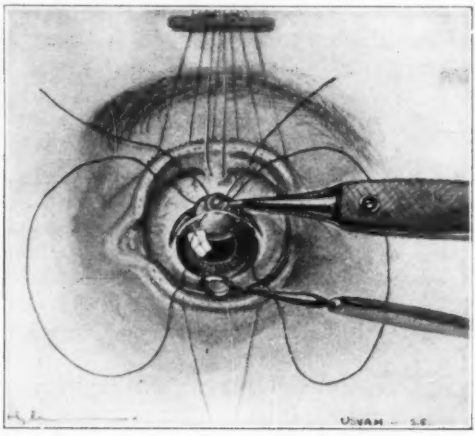

Fig. 4.—Verhoeff extraction with head-first delivery of the lens by traction and pressure on the limbus.

to bulge against the zonular membrane, which was thus placed under definite tension (fig. 5 *A*). If pressure was applied at the opposite limbus and directed backward, the hyaloid membrane and zonule in the area of iridodialysis were seen to bulge slightly, but if the same pressure was moved downward onto the sclera on the side opposite the iris opening, little or no effect was seen in the portion of the vitreous visible beneath the iridodialysis opening.

These findings were demonstrated even more clearly by gonioscopic observation and by measurement of the pressure by means of the ocular dynamometer of Bailliart. In the gonioscopic view of the angle of the anterior chamber, one could see the ciliary processes through the iridodi-

alysis opening, but only a very small portion of the origin of the zonular bundle was visible. When the 6 mm. foot plate of the ocular dynamometer was applied to the sclera in the region of the ora serrata on the same side as the area of iridodialysis and gradually increasing pressure was directed, as though to "pinch off and lift" the vitreous body, the zonular bundles suddenly appeared to bulge slightly forward into the iris opening,

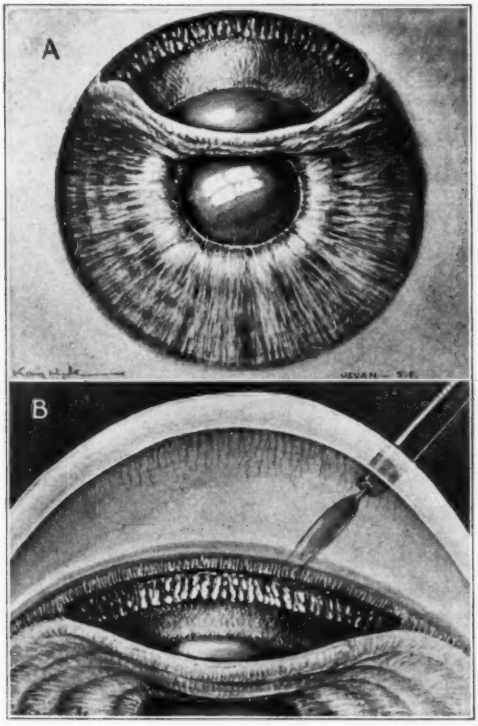

Fig. 5.—*A*, zonular tension produced by a wedge of vitreous in the iridodialysis opening, with pressure applied to the sclera on the same side 8 mm. from the limbus. *B*, gonioscopic view of production of zonular tension by a vitreous wedge in the iridodialysis opening. Pressure on the sclera was applied 8 to 10 mm. from the limbus.

and almost the entire length of the zonule became visible (fig. 5 *B*). The pressure required to demonstrate this movement was 50 Gm. Similar results have recently been demonstrated, though to a lesser extent, in a case of congenital coloboma of the iris. Even in a normal

eye, especially in a person over 50 years of age and with a low normal intraocular pressure, one can demonstrate in certain cases a slight, but definite, forward bulge of the iris in the depths of the angle under the same conditions of application of pressure (fig. 6).

Later, when an attempt was made to repair the iridodialysis defect a large keratome incision was made at the corneal limbus, thus simulating the conditions in an eye opened for cataract extraction. With the absence of an anterior chamber and with lowered intraocular pressure, it required only 20 Gm. of scleral pressure on the same side to show the forward movement of the vitreous and zonular bundles. The same amount of pressure on the opposite corneal limbus also produced tension in the visible zonular bundles, but it required 40 Gm. when applied to the opposite sclera in the region of the ora serrata to lift the entire vitreous sufficiently that movement of the zonule could be seen.

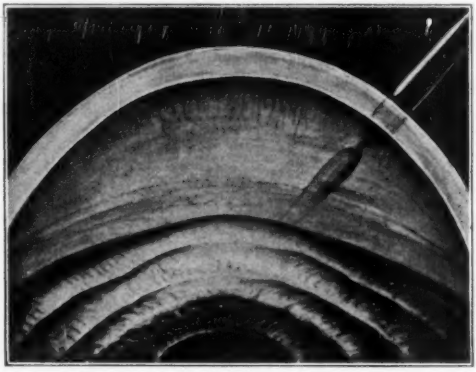

Fig. 6.—Gonioscopic view of production of a bulge of the iris by low scleral pressure. The bulge is shown as slightly exaggerated.

If an eye on which an intracapsular extraction with round pupil and peripheral iridotomy has been performed is observed with the slit lamp under these conditions of application of pressure, the movement of the vitreous in the pupillary area can be seen to follow the same general pattern, i. e., forward into the lower part of the pupil when the pressure is applied low on the sclera and across the pupillary area into its upper part when pressure is on the limbus.

These clinical observations on living human eyes were of sufficient interest to stimulate simple experiments on enucleated animal eyes, and later on human cadaver eyes. The experiments differed from those of Goldsmith [5] mainly in the maintenance of an intact vitreous with zonular rupture produced by pressure. Capsular traction with the

Sinclair, Arruga or Verhoeff forceps was reduced to an amount barely necessary to guide the direction of rotation of the lens in its escape from negative pressure of the hyaloid fossa. The eresiphake was not used, as it was in Goldsmith's work. I believe that the mechanism of its action in zonular rupture is based on an entirely different basic principle, and is deserving of a separate study, already begun.

Enucleated pig eyes were studied after corneal section with and without iridectomy, after removal of the cornea with and without iridectomy, after removal of the cornea and sclera as far back as the ora serrata and after complete removal of the corneoscleral coat, with only the uveal sac, containing the retina, vitreous and lens, remaining. The reaction of these enucleated pig eyes to the applications of pressure previously outlined was exactly the same as that in living human eyes except that much greater degrees of pressure were required to produce the same effect. This difference might have been expected because of the greater solidity of the vitreous, the tougher zonular membrane and the more rigid iris tissue. It was virtually impossible to perform an intracapsular extraction through a round pupil, and rupture of the zonule by traction on the capsule was also extremely difficult. Rupture of the zonule with dislocation and complete extraction of the lens was accomplished on many eyes by the tumbling method, after complete iridectomy by pressure applied low on the sclera with herniation of the vitreous through the lower zonular bundles, but was nearly impossible with the Verhoeff maneuver, owing to the almost invariable prolapse of vitreous ahead of the lens. Because of the solid nature of the vitreous body in the pig's eye, extreme degrees of pressure were required to simulate loss of vitreous, so that conditions in these eyes could never be considered analogous to those existing in human eyes.

Extractions on rabbit eyes were entirely unsatisfactory, owing to the enormous relative size of the lens, the fragility of the zonule and the tendency to sudden total dislocation of the lens, whether accomplished by traction or by pressure or by both.

Observations on kitten eyes were quite satisfactory and agreed closely with the findings in human cadaver eyes.

The most interesting anatomic studies were those on fresh human cadaver eyes, both *in situ* and after enucleation. Six of these eyes were examined, 2 *in situ* and 4 after enucleation. One of the enucleated eyes was used with the cornea in place. After a Graefe section, a peripheral iridotomy was performed. The lips of the wound were held open about 3 mm. Gradually increasing pressure was applied to the lower part of the corneal limbus. It required only 15 to 20 Gm. of pressure to produce a definite forward tilting of the superior edge of the lens, with slight bulging of the iris and noticeable stretching of the iridotomy opening.

The area of pressure was shifted to the sclera 6 to 8 mm. below the limbus, the direction of pressure being upward and slightly forward, as though to lift the eye from the orbit. The globe was soft, so that a pressure of 30 Gm. produced a noticeable scleral dimple, but it required only 20 Gm. of pressure to produce a forward tilt of the lower edge of the lens and a bulge in the lower part of the iris (fig. 7). Further pressure, up to 40 Gm., was required to produce sudden rupture of the lower zonular bundles and forward and upward dislocation of the lens. When the scleral pressure was released, the lens settled back into place. On resumption of low scleral pressure, the vitreous presented in the lower part of the pupil, this time tilting the lens only slightly on its horizontal axis. With maintenance of this herniation of the vitreous and with the point of application slowly moved nearer the limbus, the vitreous body could be made slowly, to tumble the lens so that its inferior edge moved upward into the wound. By this time the pressure was entirely

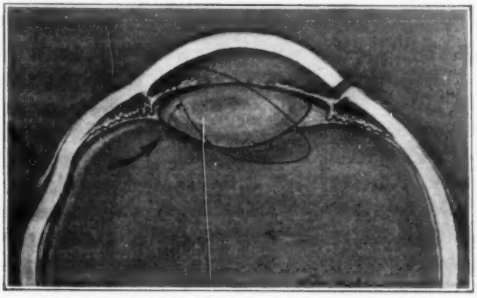

Fig. 7.—Direction of movement of the vitreous and rotation of the lens with pressure on the sclera 10 mm. from the limbus.

corneal, and final extraction of the lens was accomplished by upward massage of the cornea. This maneuver is apparently very similar to the Smith-Indian extraction. At no time during the procedure did there seem to be any great danger of prolapse of the vitreous, even though the dislocation and extraction were interrupted. If the sudden rupture of the zonule, with forward tilting of the lower edge of the lens, had been followed by a quick shift of pressure upward onto the limbus, it is felt that even less tendency to herniation of the vitreous would have been noted.

The second enucleated eye was prepared by complete removal of the cornea, and a full iridectomy was performed. With a pressure of 15 Gm. on the lower limbal area, the superior edge of the lens was tilted forward and grasped by the Verhoeff capsule forceps which straddled the equator in the area of the iridectomy. This ruptured the superior

zonular lamella. Further pressure up to 30 Gm., caused the vitreous to present in the area of the iridectomy, ahead of the lens. When the pressure was shifted downward on the sclera, the presentation of the vitreous above was lessened, but attempts to make it produce rupture of the lower zonular fibers were only partially successful, and the extraction of the lens was accomplished mainly by traction on the capsule, assisted by pressure from below while the posterior surface of the lens was held against the scleral edge of the wound to prevent the upward prolapse of the vitreous. This, as mentioned before, is the deliberate maneuver of the Verhoeff method of extraction.

The third enucleated eye was utilized to demonstrate in an exaggerated fashion the action of the wedge of vitreous in rupture of the zonule and dislocation of the lens in the tumbling method of extraction with both capsular traction and external pressure.

A Graefe section of the cornea was made, after which the lateral half of the cornea was removed and the medial half of the incision was somewhat enlarged. A band of sclera 6 mm. wide was dissected from the underlying ciliary body from 9 to 3 o'clock. A small peripheral iridotomy was performed. A very small bite of the anterior capsule of the lens was grasped at the lower pupillary border by the Sinclair [6] cross action forceps, and barely enough traction was exerted to place the lower part of the zonule under slight tension. When pressure was applied at the lower limbus with the foot plate of the ocular dynamometer, or with the edge or flat surface of the Verhoeff ring, the upward bulge of the vitreous was clearly shown in the area of the ciliary body above where its scleral support had been removed. When the same amount of pressure was shifted downward onto the sclera, the bulge of the vitreous in the unsupported area above was noticably lessened, and, in fact, could easily be seen to have been brought to bear on the lower part of the zonule and the iris. In spite of the lack of support over the superior ciliary body, where a pressure transmitted through the vitreous could most easily be demonstrated, further pressure on the lower portion of the sclera readily produced an inferior wedge of the vitreous (fig. 8*A*), which ruptured the zonule below and dislocated the lower edge of the lens forward (fig. 8*B*). At this point slightly more traction was applied to the capsule, and with continued pressure below the lens was rotated or tumbled on its horizontal axis and freed from the negative pressure of the hyaloid fossa. Once tumbled, the lens was finally freed from its upper zonular attachment and extracted mainly by traction, but even this was assisted by moving the pressure upward into the limbal area.

6. Sinclair, A. H. H.: Intra-Capsular Extraction of Cataract, Tr. Ophth. Soc. U. Kingdom **52**:57, 1933.

The fourth enucleated eye was used to demonstrate Kirby's [7] method of head-first extraction of the lens after rupture of the inferior part of the zonule by direct point pressure on the taut zonule without utilizing the vitreal wedge.

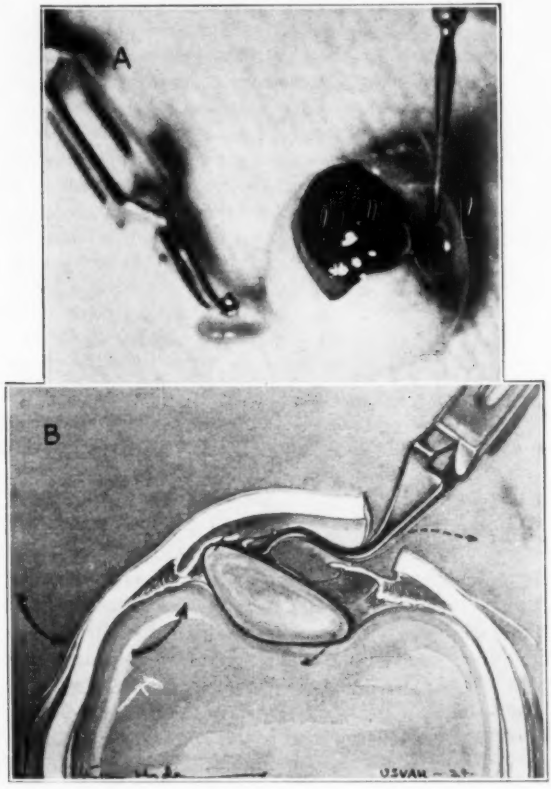

Fig. 8.—*A*, bulge of vitreous produced beneath the inferior portion of the iris by low pressure on the sclera in spite of removal of scleral support above. *B*, direction of movement of the vitreous and rotation of the lens in extraction by combined pressure and traction, with pressure exerted 8 to 10 mm. from the limbus.

7. Kirby, D. B.: The Development of a System of Intra-Capsular Cataract Extraction, Am. J. Ophth. **27**:124 (Feb.) 1944.

Kirby's detailed instructions as to traction at 12 o'clock combined with point pressure at 6 o'clock directly over the tensed zonule, rotation of traction to 2 o'clock combined with point pressure at 8 o'clock and rotation to 10 o'clock combined with point pressure at 4 o'clock were carefully followed. The zonule ruptured readily below, and further upward traction slid the lens head first through the wound.

Several things with reference to the action of the vitreous were noted during the procedure. Without a full iridectomy it was necessary to lift the superior edge of the lens so that on rupture of the lower part of the zonule the lens would not engage the iris above. When this was done and point pressure was applied on the lower limbus sufficient to rupture the zonule below, there was a rather pronounced bulging of the vitreous in the superior zonular area, as had been demonstrated in previous experiments. The same observation was made on 2 kitten eyes and in 1 case of extraction in the living human eye. In the latter, a full iridectomy was performed, and it is my belief that the rupture of the superior zonular area occurred through the action of the vitreous wedge, while the rupture of the inferior zonular fibers was accomplished by direct pressure. The extraction of the lens was accomplished almost wholly by traction.

The 2 human cadaver eyes which were operated on *in situ* were utilized primarily to demonstrate the mechanism of occurrence of certain operative and postoperative complications. Some of the accessory steps in intracapsular cataract extraction and their bearing on these complications will now be discussed in the light of these experiments.

ANESTHESIA AND LID CONTROL

The use of retrobulbar injections and akinesis has become so well established as to need little comment. It is practically universal, and the relative merits of the infiltration and the O'Brien [8] nerve block methods are largely a matter of the operator's personal preference. In certain prominent eyes in which there may be more than the usual danger of globe compression, it is probably wise to use both methods. It is probable that the O'Brien method gives more complete paralysis when successfully applied. On the other hand, infiltration has the advantage of producing anesthesia of the skin, which may be a valuable aid in preventing sudden movement of the patient.

For years the question of retraction of the lid has been a vexed one. Many types of speculums have been devised, but all have the disadvantage of introducing a hard metal surface between the lid and the

8. O'Brien, C. S.: Akinesia During Cataract Extraction, Arch. Ophth. **1**:447 (April) 1929.

globe and of not being quickly removable. Older operators have insisted on the importance of lid control and have preferred retractors, hand held by expert assistants.

When Horner [9] introduced his method of lid control by sutures (fig. 9), he was attempting to find a method which would be safe in the hands of untrained assistants. This it has proved to be, as judged by

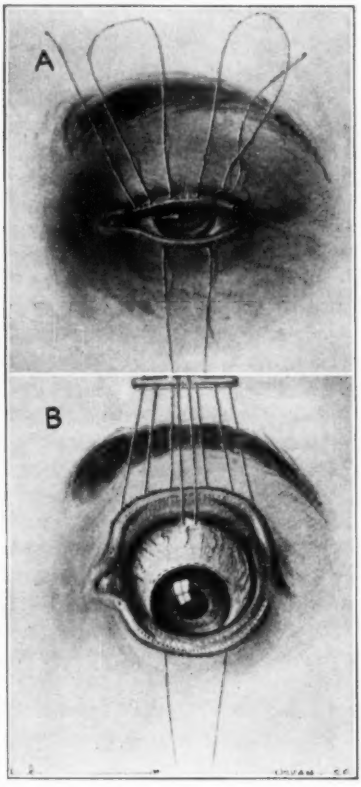

Fig. 9.—*A*, Horner lid sutures. *B*, Horner method of retraction of the lid with sutures.

experience with it at the University of California since 1931. It is felt that many potential operative complications have been avoided, and a

9. Horner, W. D.: Sutures for Lid Control in Cataract Operations, Am. J. Ophth. **18**:33 (Jan.) 1935.

few serious complications minimized, by the quick lifting closure of the lids, which is possible only with this method of lid control.

In the cadaver eyes operated on *in situ*, it was found that even the simplest type of spring speculum required an appreciable time for its removal, that the more complicated the speculum the more difficult it was to handle and that in the presence of attempted lid closure (simulated by a purse string suture in the lid margin) the blades of the speculum produced added pressure on the globe. The lower blade of the speculum exerted its pressure at the corneal limbus, when the eye was rolled upward, in exactly the position to give maximum transmitted pressure through the vitreous into the area of the corneal wound.

The suture method of lid control had the drawback that, under too much tension, the upper lid tended to evert, causing the upper edge of the tarsal plate to indent the globe well back on the sclera in a position to produce presentation of the vitreous in the wound area. This tendency to eversion was not increased by attempted closure of the lids and could be avoided by not lifting too much on the suture. In lax-lidded persons, it might prove a source of trouble.

In the study of possible sources of postoperative wound rupture, prolapse of the iris and prolapse of the vitreous after intracapsular cataract extraction, the positions of the lids were noted on living human subjects under conditions of relaxation, moderate lid closure and tight lid closure, and attempts were made to simulate these positions in the cadaver. It was found that when the eye was closed the lid margins exerted a variable pressure on the globe at the lower corneal limbus, or merely on the lower part of the cornea. Even when the upper lid was eliminated as a source of trouble, the sharp margin of the lower lid was capable of exerting considerable linear pressure at or near the limbus in exactly the position where it would produce maximum vitreous pressure in the wound area. When this pressure was simulated either directly or by artificial tautness in the lower lid, the wound could be made to gape on either side of a corneoscleral suture, and even the slight pressure of 15 Gm. caused a movement of the iris toward the wound.

A conjunctival flap offered little resistance to this pressure. A single McLean [10] type of suture allowed prolapse of the iris at 10 and 2 o'clock. A modified Stallard [11] or mattress type of corneoscleral suture offered slightly more resistance, owing to its broader base. The most adequate protection against a measured or constant pressure up to 35 Gm. was offered by three corneoscleral sutures of McLean type or by a rather

10. McLean, J. M.: New Corneoscleral Suture, Arch. Ophth. **23**:554 (March) 1940.

11. Stallard, H. B.: Corneo-Scleral Suture in Cataract Extraction, Brit. J. Ophth. **22**:269, 1938.

wide mattress type of corneoscleral suture (fig. 10) combined with a Van Lint [12] type of conjunctival flap, pulled down from above.

IRIDECTOMY

It is fairly generally agreed that the preservation of the round pupil in intracapsular cataract extraction is much to be desired. In addition to its optical and cosmetic advantages, its greatest value, as pointed out by Knapp, lies in its action as a barrier to prolapse of the vitreous. Manipulation of the cadaver eye *in situ* showed that it required almost twice as much pressure at the lower corneal limbus to force a prolapse of the intact vitreous past an intact iris, or one on which a small peripheral iridotomy had been performed, as that required after a full iridectomy. Knapp [1] raised the objection that a peripheral iridotomy

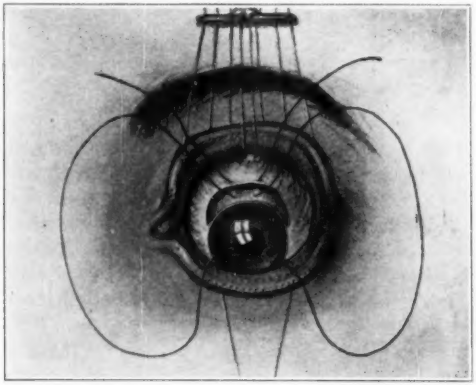

Fig. 10.—Corneoscleral mattress suture and conjunctival flap.

is difficult to perform and that the possibility of cutting into the vitreous is always present. If the bite of the iris forceps is kept small and the root of the iris is tented before cutting, this can be avoided (fig. 11). The special bayonet iris forceps of Traquair [13] has a set screw, or wheel, between its blades, which may be preset at any desired width and will insure a small bite at the jaws.

In the cadaver eye a deliberate injury to the vitreous by a roughly performed peripheral iridotomy so changed the character of the vitreous that only one-third as much pressure was required to produce its pro-

12. Van Lint: Astigmatisme post-opératoire dans l'extraction de cataracte avec glissement de la conjonctive, Ann. d'ocul. **151**:418, 1914.

13. Traquair, H. M.: Personal communication to the author.

lapse after injury as before. The effect of a small nick in the hyaloid membrane is analogous to that of nicking the edge of a case-hardened glass lens. It would almost appear that the entire character of the vitreous body had been changed, so that it became semifluid and almost ceased to follow the previously outlined rules of pressure transmission. Even the slightest pressure at any point on the globe, whether near the limbus or on the sclera, produced immediate presentation and prolapse of vitreous, whereas before this injury the pressure was transmitted through the vitreous, as outlined earlier in this paper.

It is suggested that in cataract extractions in the presence of known fluid or injured vitreous pressure be applied to the globe with extreme caution and that extraction be accomplished mainly by traction.

In the presence of normal vitreous it could not be demonstrated experimentally on the cadaver eye that a double iridotomy was any more

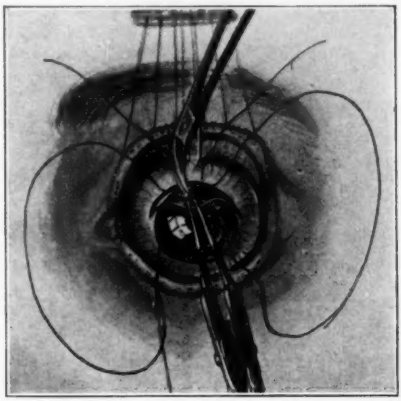

Fig. 11.—Peripheral iridotomy with small iris bite and tenting of the iris.

effective in preventing prolapse of the iris than a single one. It must be admitted, however, that these prolapses were produced by vitreous pressure rather than by sudden leakage of aqueous, such as might produce a prolapse of iris in a living eye on the fourth or the fifth postoperative day.

EXTRACTION BY TRACTION

Except for zonular rupture and lens extraction by phakoeresis, which involves an entirely different basic principle from that under discussion here, it is my considered opinion that zonular rupture and total extraction by capsular traction alone are very difficult and extremely uncertain. Even allowing for the known fragility of the zonule in the presence of mature cataract in the aged person, the percentage of capsular ruptures

is very high when attempts are made to extract the lens by traction alone. For this reason, most experienced operators combine traction with external pressure. On the other hand, Arruga [14] stated that he depended mainly on traction, and the nature of the Verhoeff [3] method demands preponderance of traction over pressure.

Aside from any other consideration, such as rupture of the hyaloid membrane, prolapse of the vitreous or injury to the ciliary processes, cited as disadvantages of extraction by pressure, the higher the ratio of traction to pressure, the greater the likelihood of rupture of the capsule.

INJECTION OF AIR INTO THE ANTERIOR CHAMBER

Ever since MacMillan [15] advocated the injection of air under the conjunctival flap and into the anterior chamber in trephinations for

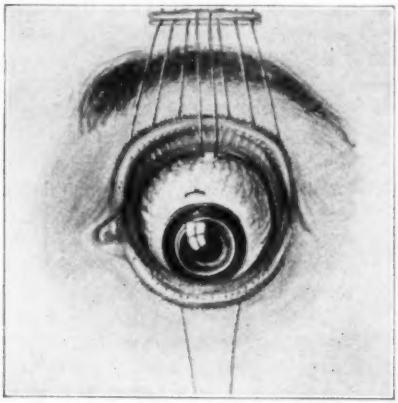

Fig. 12.—Corneoscleral suture tied and air bubble in the anterior chamber.

glaucoma, I have made this procedure routine after intracapsular cataract extraction. Besides aiding in the more rapid and even reformation of the anterior chamber, it also acts as a "cushion" to prevent prolapse of the iris in the event of postoperative pressure of the lid margins on the cornea. It was repeatedly demonstrated on cadaver eyes and on a living human eye that a pressure of 30 Gm. on the lower corneal limbus would produce a small prolapse of the iris if the anterior chamber was absent or was partially filled with fluid. If an air bubble was introduced

14. Arruga, H.: Les détails techniques de l'extraction intracapsulaire du cristallin, Bull. et mém. Soc. franc. d'opht. **46**:270, 1933.

15. MacMillan, J. A.: Injection of Air as Factor in Maintaining Filtration After Corneoscleral Trephining in Glaucoma, Arch. Ophth. **22**:968 (Dec.) 1939.

into the anterior chamber and a similar pressure was applied to the globe, the air escaped in a rush, but the iris did not prolapse into the wound. Only one precaution is necessary. If too much air is injected into the anterior chamber, producing an abnormally deep chamber, it is possible for some of the air to enter the posterior chamber and become trapped behind the iris. The application of external pressure then produces a prolapse of the iris, with the exit of air through the wound.

SUMMARY AND CONCLUSIONS

It is fairly generally agreed among ophthalmologists that the ideal cataract extraction from the point of view both of the patient and of the surgeon is that performed by the intracapsular method through the intact pupil, with employment of all the modern safety measures of proved merit.

In order to acquire the greatest proficiency in intracapsular cataract extraction, the surgeon should have an understanding of the physical principles underlying the dislocation and delivery of the lens.

It can be demonstrated by slit lamp microscopy, by gonioscopic observation, during actual intracapsular cataract extraction in the living eye, on animal eyes and on human cadaver eyes that the rupture of the zonular lamella of the lens is accomplished primarily by the production by external pressure of a wedge of vitreous, which is made to insert itself between the equator of the lens and the ciliary processes. This deformity of the vitreous can be produced at whatever site is desired, depending on the method and the point of application of the external pressure and on whether or not one wishes to tumble or to slide the lens from its position in the hyaloid fossa.

The normal vitreous is a tissue and cannot be considered as a fluid, in which pressure at one point is transmitted equally to all other parts.

Capsular traction facilitates dislocation of the lens by stretching the zonule, but does not normally produce its rupture.

Certain factors of safety of utmost importance in intracapsular cataract extraction may be reevaluated in the light of these basic physical principles.

384 Post Street.

NONSYPHILITIC INTERSTITIAL KERATITIS WITH VESTIBULOAUDITORY SYMPTOMS

Report of Four Additional Cases

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BOSTON

IN 1945 I described 4 patients presenting a syndrome consisting of a characteristic type of interstitial keratitis and vestibuloauditory symptoms like those of Ménière's disease and resulting, usually, in complete deafness.[1] Only 1 previous instance, and this one questionable, could be found from an examination of the literature. The ocular signs consisted early of a granular type of corneal infiltrate, patchy in distribution, situated predominantly in the posterior half of the cornea; late the cornea became vascularized. The keratitis was bilateral in 3 of the 4 patients and ran a characteristically chronic course, varying in its severity from day to day and from eye to eye. There was little or no reaction in the anterior chamber or the iris. The vestibuloauditory symptoms, coming on within a few days of the ocular symptoms, consisted typically of severe vertigo, tinnitus, nystagmus and rapidly progressive deafness. There was an elevated white blood cell count, and usually a mild eosinophilia, but no other evidence of systemic disease. The type of manifestations, the course of the disease, the absence of a family history and the consistently negative serologic reactions were thought sufficient evidence to rule out congenital syphilis as the cause. All the patients were young adults.

It is the purpose of the present communication to report 4 additional patients, 3 of whom were observed by me and 1 by Dr. Francis Keil. Chief interest attaches to the first patient, in whom, despite several suggestive leads, exhaustive investigation failed to disclose the cause. The fourth patient is also of interest, inasmuch as the deafness cleared up entirely.

REPORT OF CASES

CASE 1.—*History.*—F. D., an American-born Italian, aged 24, had been under observation fourteen months. He is the second of five children, all of whom are living and well. According to the parents, both of whom are living and well, there were no miscarriages or stillbirths in the family or evidence of venereal

From the Howe Laboratory of Ophthalmology, Harvard Medical School, and the Massachusetts Eye and Ear Infirmary.

1. Cogan, D. G.: Syndrome of Nonsyphilitic Interstitial Keratitis and Vestibuloauditory Symptoms, Arch. Ophth. **33**:144-149 (Feb.) 1945.

disease. Hinton tests of the parents' blood gave negative reactions. There was no history of significant ocular, aural or systemic disease in the family.

The noteworthy medical events in the patient's history were the presence of cervical lymphadenopathy in childhood and an episode of what was believed to be rheumatic fever two years prior to the present illness. This episode was characterized by sore throat, from which streptococci were cultured, followed by extensive "muscle pains," lasting about one week. He was treated at this time by sulfonamide compounds, but not by salicylates. He was subsequently found to have an enlargement of the heart and a grade 3 systolic murmur, which were attributed to this episode. Eight months prior to the onset of the present illness, while the patient was attached to the Navy in the vicinity of Japan, he lost approximately 30 pounds (13.6 Kg.) in weight over a period of three to four months, without apparent cause. He subsequently received a routine discharge from the Navy, no medical abnormality being noted. One month prior to the onset of the present illness, he had for a few days a sore throat, stiff neck and swollen glands. This condition was not treated by sulfonamide drugs.

Present Illness.—The present illness began, fourteen months ago, with redness of the right eye, tenderness and photophobia. Three days later, when the ocular condition appeared to be improving, severe bilateral tinnitus developed, and the patient became so dizzy that he was forced to stay in bed, acutely ill. Within one week the vertigo improved, but the patient had become profoundly deaf, first in the left ear and then in the right. During the first week, also, the left eye became inflamed, like the right eye. Beginning five days after onset, the patient was given intravenous injections of penicillin, in doses of 300,000 units daily. On the fifteenth day this medication was changed to streptomycin, one injection daily (dose not known) but penicillin therapy was reestablished on the nineteenth day, when a rash developed.

The examination to be reported took place one month after the onset of the present illness.

Physical Examination.—Except for signs referable to the oculoaural systems, no abnormality was noted on physical or neurologic examination.

Ophthalmologic Examination.—Right Eye: Vision was 20/30, with moderate ciliary redness. The cornea contained 20 to 30 ill defined, round and confluent opacities, situated in the midstroma and the posterior third of the cornea. These opacities were scattered throughout the cornea, showing no preferential involvement of the axial or limbal portion. Between the opacities the cornea was grossly clear. The opacities, which were yellowish and granular, were interpreted as infiltrate. The anterior surface of the cornea was smooth and lustrous; the posterior surface showed no folds, irregularities or deposits. Corneal sensitivity was normal. The anterior chamber was clear, showing no cells or outstanding beam. The pupil dilated well with atropine and showed no synechia. The fundus was seen fairly well and appeared normal.

Left Eye: Vision was 20/20. The findings in this eye were the same as those in the right except that at this examination they were less pronounced, the corneal infiltrates being visible only with a bright light.

Ocular motility was normal except that nystagmus was present to a somewhat greater extent than normal on conjugate gaze to either side.

Otologic Examination.—No nystagmus was induced by irrigating either external auditory meatus with 200 cc. of cold alcohol or by rotating the patient in a Bárány chair with the head placed in the usual position, nor did the patient have any subjective sense of being rotated. The deafness was complete.

Neurologic Examination.—No abnormality was found except for deafness.

Laboratory Data.—The urine was normal. Studies of the blood made repeatedly over a period of one month were reported as follows: The red cell count was more than 5,000,000 on several occasions. The white blood cell count (made four times) averaged 10,650 per cubic millimeter. The average differential white cell count, made seven times, was: polymorphonuclear cells, 56 per cent; lymphocytes, 26 per cent; monocytes, 6 per cent, and eosinophils, 12 per cent (with a variation of from 5 to 28 per cent). The sedimentation rates ranged from 1.3 to 1.5 mm. per minute, with corresponding hematocrit readings of 43 to 41 per cent. Serum agglutination tests were negative for brucellosis, typhoid and paratyphoid A and B. Wassermann reactions of the blood were negative. A smear from the conjunctiva showed no eosinophils, bacteria or inclusion bodies. Culture of material taken from the conjunctiva showed coagulase-positive Staphylococcus aureus on one occasion, but no growth on two occasions. Lumbar puncture showed normal dynamics, with a total protein content of 21 mg. per hundred cubic centimeters and a flat colloidal gold curve. The Wassermann reaction of the spinal fluid was negative. A specimen of the stool was negative for cysts. Intradermal tests with the common allergens, including molds, showed no significant sensitivity. A roentgenogram of the chest revealed nothing abnormal. Injection of serum from the patient into the abdomen of a guinea pig was without apparent effect on the guinea pig. Biopsy of tissue from the deltoid muscle showed no evidence of trichinosis, and injection into a guinea pig of material from one of the patient's cervical glands failed to produce tuberculosis. The gland itself, however, showed chronic inflammation and calcification compatible with old tuberculosis.

Course of Illness.—The patient was studied in the hospital for one month, during which time most of the foregoing laboratory data were obtained. The patient's temperature was at no time elevated, nor was there evidence, other than that noted in the blood studies, to suggest systemic disease.

The ocular disease continued to be characterized by patchy infiltrate in the deep corneal stroma but showed an extraordinary variation from day to day and from one eye to the other. At times one eye would show considerable infiltrate, with corresponding congestion of the globe, but these signs often cleared dramatically overnight, only to become manifest in the other eye. Yet the corneas showed no pronounced swelling and the anterior chambers no more than a faint beam, and only rarely were keratic precipitates to be found on the posterior surface of the cornea. Vascularization of the deep cornea, observed only after the disease had been present for four months, was never a conspicuous feature. Seven months after onset, the red blood cell count was 4,380,000 and the white blood cell count 12,400, with 3 per cent eosinophils. The sedimentation rate was 46 mm. in one hour. At the end of fourteen months the ocular disease was still active and continued to show its characteristic fluctuations, but at less frequent intervals.

The otologic signs, which were characterized by an incapacitating vertigo and tinnitus at the onset, consisted finally in complete deafness.

In addition to the local treatment to the eyes, the patient was given a transfusion of plasma (200 cc.) from a patient who had had the same disease one year previously. No effect on the course of the patient's ocular condition was noted. Penicillin given systemically at the onset of the disease was without benefit. After the disease had been present for eleven months, the patient was given systemically a course of riboflavin, nicotinamide and thiamine hydrochloride [2] with subsequent improvement in the eyes.

2. This treatment was suggested and supervised by Dr. Miles Atkinson.

Résumé.—In a young man with a history of cervical lymphadenopathy and rheumatic fever, there developed within the course of a few days bilateral interstitial keratitis, tinnitus and profound vertigo. The keratitis was characterized by a granular infiltrate, arranged in a patchy distribution, situated in the deep stroma. Showing considerable variation in severity from day to day, the keratitis persisted about fourteen months and the corneas became vascularized, but at no time was the process severe enough to impair vision seriously. The initial tinnitus and vertigo disappeared within the first month as total deafness supervened and as the labyrinths became inactive. The only abnormal laboratory findings were an elevated white blood cell count (average, 10,650), eosinophilia, with a count varying from 5 to 28 per cent, and an increased sedimentation rate, 1.3 to 1.5 mm. per minute. There was no evidence of congenital or acquired syphilis. Treatment with penicillin and by transfusion of convalescent serum was without benefit, but the patient improved after multiple vitamin therapy.

CASE 2.—*History.*—R. E., a native-born Guatemalan aged 20 years, was examined fifteen months after the onset of his present illness. He is the second of 3 siblings, all of whom, as well as the father and mother, are living and well. He denied having had systemic illness other than childhood diseases, but one week prior to the onset of the present illness he had been vaccinated for smallpox.[3] He had not received streptomycin either during or prior to the present illness.

Present Illness.—The onset was characterized by tinnitus (first in the right ear, and within a few days in the left ear) and profound vertigo, accompanied with illusory movement of the environment. Deafness was first complained of three days after the onset and progressed in four months to loss of all useful hearing. The ocular symptoms, beginning five months after the onset of the aural symptoms and two weeks after an appendectomy, were characterized, according to the patient, by pain, photophobia and redness of the right eye, but no great loss of vision. The patient was examined at this time at the Mayo Clinic, with the following findings: ". . . Three pinhead-sized areas of keratitis were present on the right cornea. Two days later the keratitis was definitely worse, and the entire cornea was milky in appearance . . . the left eye remained unaffected."

From the onset to the time of the present examination, fifteen months later, the right eye continued to be intermittently inflamed and painful. The left eye had always been normal. At the Mayo Clinic the patient was treated for one and one-half months with daily injections of vitamin B preparations and for five months with intravenous injections of histamine. There was some, although slight, recovery of hearing after four months of the latter treatment.

The patient's status ten months after onset of the ocular symptoms, and fifteen months after the onset of the vestibuloauditory symptoms, is described.

Ophthalmologic Examination.—Right Eye: Moderate redness of the ciliary body was present. The cornea showed an arcuate opacity corresponding approximately to the border of the pupil and densest in the upper portion (11 to 2 o'clock)

3. Since this report was submitted for publication, a description of a similar case has appeared, in which the disease also occurred one week after smallpox vaccination (Rosen, E.: Interstitial Keratitis and Vestibuloauditory Symptoms Following Vaccination, Arch. Ophth. **41**:24-31 [Jan.] 1949).

and in a small segment below (6 o'clock). With the biomicroscope, this opacity appeared densely granular, and was interpreted as infiltrate, with glistening particles, interpreted as cholesterol crystals. There was also a granular infiltrate of the rest of the cornea, but this was not sufficiently dense to be visible grossly. The opacities were situated predominantly in the posterior layers. The periphery of the cornea was the clearest portion. Vessels extended into the corneal stroma in its middle and posterior layers. The cornea was little, if at all, thickened, and the anterior chamber was clear. The media and fundus were normal.

Left Eye: The eye was entirely normal.

Examination of Ears, Nose and Throat (Dr. D. K. Lewis).—The nose and throat were entirely normal. There was no vestibular response with massive caloric irrigation of either ear and no response to rotation in the Bárány chair. The audiogram showed profound loss on the left with inability to comprehend speech and a hearing loss on the right averaging 60 to 65 decibels. Bone conduction was less than 50 decibels in each ear.

Laboratory Data.—The Wassermann and Hinton tests of the blood gave negative reactions. The blood smear showed a differential count as follows: polymorphonuclear cells, 66 per cent; lymphocytes, 27 per cent; monocytes, 3 per cent; eosinophils, 4 per cent; basophils, 0.

Résumé.—In a young man tinnitus and profound vertigo culminated in practically complete deafness and inactive labyrinths. Five months after onset of the aural symptoms, an inflammation developed in one eye which was recurrent for at least the next ten months. At this time there was noted in this eye a granular infiltrate in the deep cornea, having a patchy distribution and accompanied with new blood vessels and scintillating particles, thought to be cholesterol crystals. The laboratory findings were normal. There was no evidence of congenital or acquired syphilis.

Case 3 (patient of Dr. Francis C. Keil).—*History.*—An American woman, aged 20, was examined two weeks after the onset of the present illness. The patient is the youngest of 4 siblings, all of whom, as well as the parents, are living and well. There had been no stillbirths in the family, and Wassermann tests of the parents' blood gave negative reactions. The patient herself had always been well.

Present Illness.—Beginning two weeks prior to the present examination, the patient experienced a . inflammation of her eyes and a vertigo which was so severe that she could not stand unaided. She rapidly became deaf.

Physical Examination.—No abnormality was found. There were no stigmas of congenital syphilis. The roentgenogram of the chest was normal.

Ophthalmologic Examination.—Vision was 20/20 in the right eye and 20/40 in the left eye.

Both Eyes: There were marked conjunctival and slight ciliary congestion. There were many grayish white infiltrates in the posterior half of the corneal stroma. The anterior stroma and surface of the cornea appeared normal. No cells were seen in the anterior chamber, but there was a slight aqueous flare.

Otologic Examination.—An audiogram showed almost complete deafness in the higher tone ranges.

Laboratory Data.—Wassermann and Kahn reactions of the blood and spinal fluid were negative. The spinal fluid showed normal globulin and sugar contents, a normal colloidol gold curve and a cell count of 5 per cubic millimeter. The

red blood cell count was 4,500,000 and the white blood cell count was 10,200, with a distribution as follows: polymorphonuclear type, 73 per cent; lymphocytes, 25 per cent, and monocytes, 2 per cent. The Mantoux test gave a negative reaction to tuberculin.

Course of Illness.—The patient received penicillin (160,000 units daily) for ten days, with little improvement. She could not be followed further, but eight months later, by telephone report, the eyes were said to have improved somewhat, although she continued to be deaf.

Résumé.—In a young woman there developed within a two week period bilateral interstitial keratitis and profound vertigo. The interstitial keratitis has been chronic but was not accompanied with any considerable decrease in vision. The vestibuloauditory symptoms culminated in deafness. There was no evidence of syphilis in the patient or in her family. The laboratory findings were normal except for a slightly elevated white blood cell count. The condition was little improved by systemic treatment with penicillin.

CASE 4.[4]—*History.*—J. G., an American professional orchestra player, aged 30, at the time of this report had been under observation seven months. He was the first-born and only surviving sibling, the others having died in infancy (ages 1 to 1½) of pneumonia (possibly tuberculous). The patient's mother died of tuberculosis when the patient was 4 years of age. One step-sister was living and well except for postmastoiditis deafness. The patient's father was living and well.

The patient had always been well. However, he had had glycosuria, presumably of renal origin, since repeated blood sugar determinations and glucose tolerance tests had shown normal values. Routine Wassermann tests of the blood several years prior to the present illness gave negative reactions.

One month prior to the onset of the present illness the patient had a stiff neck, lasting a few days. This was unaccompanied with headache or evidence of cervical lymphadenopathy so far as the patient was aware. The patient had had no medication for this or any other condition prior to the onset of the present illness.

Present Illness.—The onset was characterized by redness and watering of the left eye. This was followed in a few days by severe vertigo, with illusory movement of the environment, vomiting, buzzing in the ears and progressive deafness. The last two symptoms applied to both ears but were most pronounced on the right side. Within a few days after onset the right eye became involved, and corneal opacities developed, with considerable photophobia and blepharospasm. The patient's local physician made the diagnosis of Ménière's disease. He was further studied at the Lawrence General Hospital, where a diagnosis of interstitial keratitis and labyrinthitis was made. Except in the eyes and ears, physical examination revealed nothing unusual. A lumbar puncture was done at this time, and examination of the cerebrospinal fluid showed a negative Hinton reaction, 74 mg. of sugar and 32 mg. of total protein, per hundred cubic centimeters, and 2.5 white cells per cubic millimeter. Roentgenograms of the chest and skull showed nothing abnormal.

During the first two months of the disease the watering, redness of the eyes and blurring of vision continued with moderate severity, the condition showing

4. Dr. Karl Riemer gave me the privilege of seeing and following this patient.

characteristic variations from day to day and possibly being worse in hot weather. The patient remained partially deaf, with what was described as a "marked hearing loss in the right ear and moderate loss in the left ear, of the perceptive type of deafness," but the loss of equilibrium, which had been profound at first, had improved somewhat in the interim. Examination of the eyes two months after the onset showed considerable ciliary injection of both eyes, with yellowish infiltrates, arranged in ill defined patches, in the deep stroma. Nevertheless, there were no precipitates on the posterior surface of either cornea, little swelling of the cornea and no perceptible synechias. The cornea showed beginning vascularization in a lamina immediately in front of Descemet's membrane. Treatment during the first two months had consisted of administration of sulfonamide drugs systemically at the start and continued mydriasis. The Wassermann reactions of the blood at this time were again negative.

Approximately three months after the onset, the patient's hearing suddenly returned. He awoke one morning and found he could hear normally, and this was corroborated by an audiogram. Since this time the only vestibuloauditory symptoms have been an occasional spell of vertigo with illusory movement of the environment, lasting only a few seconds. The eyes remained irritable, however, and examination showed the same objective signs as before except that the vascularization had extended farther toward the axis of the cornea. There was still no perceptible reaction in the anterior chamber. Although the Wassermann reacactions of the blood and spinal fluid were negative, the patient was given a series of injections of a bismuth preparation.

Approximately four and one-half months after the onset, the eyes began to feel less irritable, and on examination five months after the onset the corneal infiltrate was less marked, the eyes were whitening out and there was obviously much less photophobia than formerly. The ocular status at this time was characterized by deep corneal opacities, which were barely perceptible grossly, and deep, apparently quiescent, vascularization of the corneas.

Résumé.—In a young man there developed within a few days photophobia, blepharospasm, deafness and vertigo with illusory movement of the environment and vomiting. Examination of the eyes showed a deep infiltration of both corneas with a characteristic patchy distribution, culminating in vascularization of the corneas but unaccompanied with any appreciable reaction in the anterior chambers. The deafness, which was never complete, spontaneously resolved approximately three months and the eyes began to show improvement four and a half to five months, after the onset.

COMMENT

There can be little reasonable doubt that the clinical picture in the 4 cases presented here accorded with the syndrome previously described as nonsyphilitic interstitial keratitis with vestibuloauditory symptoms. They illustrate the characteristic day to day fluctuation in the severity of the ocular process, which was insufficiently emphasized in the previous report but which is a major feature distinguishing this type of interstitial keratitis from that due to congenital syphilis. Other distinguishing features of the ocular reaction in this syndrome are the patchy distribution, the relative mildness of the process, the absence of any striking intra-

ocular involvement, the maintenance of a relatively normal posterior surface of the cornea, the absence of any marked swelling of the cornea and the chronicity. The vestibuloauditory reactions are characterized by symptoms like those of Ménière's disease with rapid development of deafness; this is unlike the vestibuloauditory symptoms in congenital syphilis, in which there is a slow evolution of deafness without appreciable vertigo.

No conclusive evidence indicating the etiologic factors in this syndrome was apparent. One patient had a recent history of rheumatic fever and cervical lymphadenopathy, and 1 patient had had vaccination for smallpox one week prior to the onset of the present illness. The type of corneal infiltrate, the elevated sedimentation rate and the relative leukocytosis of the blood suggest a bacterial, rather than a virus, disease, but cultures have not been informative. The eosinophilia, which was marked in 1 of the present patients, appears to be characteristic of the entity, but there is no indication of what its significance is. The vertigo and deafness suggest a streptomycin-induced reaction, but only 1 of the patients gave a history of having received streptomycin, and this only after the onset of the disease.

Treatment of the disease in 1 patient with a single transfusion of convalescent plasma was without benefit. Prolonged treatment with intravenous injections of histamine may have had a slightly beneficial effect on the hearing of 1 patient. Treatment of 1 patient with a multiple vitamin preparation appeared to benefit the ocular reaction late in the course of the disease. Multiple vitamin therapy had not benefited other patients early in the course of the disease, and there was no evidence of systemic vitamin insufficiency in any of the patients.

SUMMARY

A description is given of 4 additional cases of what is believed to be the syndrome previously described as nonsyphilitic interstitial keratitis with vestibuloauditory symptoms. The interstitial keratitis was bilateral in 3 of the new cases and unilateral in 1 case. It was characterized by patchy, granular infiltrates in the deep stroma, which varied from day to day, unaccompanied with any conspicuous intraocular reaction. The vestibuloauditory symptoms were similar to those occurring in Ménière's disease and were followed by practically complete deafness in 3 of the patients, with complete recovery of the deafness in 1. The onsets of the ocular and of vestibuloauditory symptoms were within a few days of each other in 3 of the patients but were separated by a five month interval in 1 patient. As in the previously reported series, all 4 patients were young adults. All had a leukocytosis, 1 of whom had a significant eosinophilia. No conclusive evidence was ascertained regarding etiology or specific treatment.

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DIRECT SURGERY OF PARETIC OBLIQUE MUSCLES

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IT IS THE purpose of this paper to discuss seven years' experience with direct surgery of the underacting superior oblique muscle and eight years' experience with direct surgery of the weak inferior oblique muscle. Although the long-standing fear of oblique muscle surgery was gradually yielding to successful demonstrations of weakening operations on the inferior oblique muscle, it remained for Wheeler,[1] in 1935, to show that surgical correction of the superior oblique could be performed and that the underacting inferior oblique could be strengthened. Before his paper, and even after it, attempts to compensate for pareses of the oblique muscles were usually made on the vertical rectus muscles, if at all.[2] The influence of von Graefe's [3] frequently quoted interdiction of direct surgery of the oblique muscles was still strong.

The anatomy and physiology of the oblique muscles have been so adequately reviewed and discussed, especially in the recent papers of Fink [4] and Adler,[5] that reiteration here would be superfluous. Only choice of operation, operative technics and operative results will be discussed in this paper.

PARESIS OF THE SUPERIOR OBLIQUE MUSCLE

There are obvious objections to surgical procedures on the vertical rectus muscles of the same eye to correct the effects of weakness of the superior oblique. Weakening operations on the superior rectus may

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1. Wheeler, J. M.: Am. J. Ophth. **18**:1, 1935.

2. Landolt, E.: Am. J. Ophth. **6**:93, 1923. Snell, A. C.: Injuries of Superior Oblique Muscle, Arch. Ophth. **48**:111 (March) 1919. Banister, J. M.: Am. J. Ophth. **2**:537, 1928. Savin, L. H.: Proc. Roy. Soc. Med. **27**:1058, 1934. Jaensch, P. A.: Klin. Monatsbl. f. Augenh. **97**:807, 1936. Bielschowsky, A.: ibid. **69**:611, 1922. Dunnington, J. H.: Am. J. Ophth. **14**:1140, 1931.

3. von Graefe., A., in Graefe, A., and Saemisch, T.: Handbuch der Augenheilkunde, Leipzig, Wilhelm Engelmann, 1898, vol. 8, p. 84.

4. Fink, W. H.: Tr. Am. Acad. Ophth. **50**:269, 1946.

5. Adler, F. H.: Tr. Am. Ophth. Soc. **43**:255, 1945.

tend to neutralize the vertical discrepancy in the primary position but have two distinct disadvantages. They add to the already existing imbalance in lower nasal gaze a new and unnecessary defect in upper temporal gaze. They tend also to increase the torsional defect by weakening the other intorting muscle. Strengthening operations on the inferior rectus have similar disadvantages, calling on a lower temporal hypotropia to meet the defect of a lower nasal hypertropia and increasing the torsional defect.

Weakening of the inferior oblique of the same eye is less unsatisfactory, for it tends to balance the torsional defect; but, instead of correcting the lower nasal hypertropia, it attempts to neutralize it by superimposing an upper nasal hypotropia.

All the aforementioned procedures are in conflict with the very important and useful cardinal principle enunciated by Duane[6] and reemphasized by White,[7] that muscle surgery should be confined to the field of action of the defective muscle. In this connection, the only two satisfactory procedures are direct strengthening of the paretic superior oblique and weakening of inferior rectus of the opposite eye. Of these two approaches, the first is to be preferred, for three reasons. It attempts to restore normal relations of motor function instead of balancing an existing defect by adding a second defect. It avoids the difficulties with altered lid fissures inherent in any extensive surgical work on the vertical rectus muscles and also avoids the difficulties involved in interference with the ligament of Lockwood.[8] It tends to restore better muscle balance in the position of depressed convergence, so important in reading and other pursuits of modern life.

TECHNIC

With this reasoning in mind, my colleagues and I have been making attempts at direct strengthening of paretic superior oblique muscles since 1941. The first technic adopted was a modification of Wheeler's.[1] His technic, while described as "advancement," is not advancement in the usual sense of the term but, rather, a folding and advancement of the middle of the tendon—a compromise between advancement and tucking. Because of the fear that his technic might result in failure of permanent adhesion of the smooth tendon surface to the sclera, a true tuck was performed at the scleral insertion of the muscle in inverted fashion, with the tucked loop buried beneath the tendon (fig. 1*A*). This fear seems to have been justified by the subsequent experiences of McGuire.[9] This modified technic was successful, as will be illustrated in the case reports to follow, but appeared to have room for improvement. The amount of reaction following the extensive exposure and dissection was more than desirable. Removal and replacement of the superior rectus seemed undesirable.

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6. Duane, A., cited by White.[7]

7. White, J. W.: Tr. Pacific Coast Oto-Ophth. Soc. **26**:112, 1941.

8. Fink, W. H.: Ligament of Lockwood in Relation to Surgery of the Inferior Oblique and Inferior Rectus Muscles, Arch. Ophth. **39**:371 (March) 1948.

9. McGuire, W. P.: Tr. Am. Ophth. Soc. **44**:527, 1946.

A second method was then devised to avoid interference with the superior rectus and to reduce the amount of dissection. A conjunctival incision was started at the nasal end of the tendon of the superior rectus and carried nasally and posteriorly. With the globe turned down by a muscle hook or a traction suture placed beneath the superior rectus tendon, a hook was introduced between Tenon's capsule and the sclera and passed backward in the orbit about 1 cm., until it could be turned to engage and withdraw the tendon of the superior oblique. (This approach to the tendon has since been described by Berke [10] in his operations of tenotomy and tenectomy.) The mobolized tendon was then tucked in the usual fashion (fig. 1*B*) and allowed to retract. This method was satisfactory in my hands, but a colleague [11] reported his unfortunate experience when he placed a tuck too close to the trochlea and produced abrupt limitation of nasal depression of the eye.

With this potential difficulty in mind, a third technic, having the advantages of the first two, was devised. This method proved most satisfactory and is still in use today. Conjunctiva and Tenon's capsule are incised at the temporal border of the superior rectus tendon, and the incision is carried temporally and posteriorly. With the superior rectus tendon on a muscle hook or traction suture, the eye is turned strongly downward and nasally.

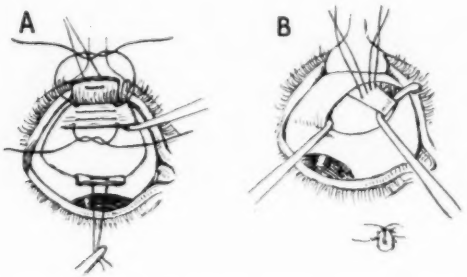


Fig. 1.—*A*, inverted tucking of superior oblique with temporary removal of superior rectus. *B*, tucking of superior oblique by nasal approach without disturbing the superior rectus.

In some eyes the insertion of the superior oblique muscle is thus exposed to direct sight. In others the belly of the superior rectus may be lightly retracted to allow direct exposure. The superior oblique tendon is then withdrawn from beneath the superior rectus, freed from the light attachments to its sheath and tucked to the desirable amount (fig. 2*A*). If desired, the apex of the tucked loop may be anchored to the sclera (fig. 2*B*). This anchoring is not designed to be the main support, as in Wheeler's [1] technic, but is intended merely to keep the loop in proper position. Chromic surgical gut, buried silk and buried nylon sutures have all been used satisfactorily. The amount of postoperative reaction is still somewhat greater than that encountered in the usual operations on the rectus muscles, but definitely less with Wheeler's method or with the first technic described. Tucking has been chosen rather than resection, because the tendon of a very paretic superior oblique muscle is often so thin and stringy that resection sutures

10. Berke, R. N.: Tr. Am. Ophth. Soc. **44**:304, 1946.

11. Personal communication to the author.

may conceivably pull out. True advancement has not been used, for the same reason, because the amount of further posterior dissection and exposure seems unnecessary and because of a reluctance to do too heavy suturing in the region of the macula.

RESULTS

In all, 19 tucking operations have been performed on 19 superior oblique muscles in 15 patients since 1941. The results have been generally satisfactory. Six were done by the technic illustrated in Figure 1 *A*, 4 by that in figure 1 *B* and 9 by that in figure 2. There were no serious complications, and the amounts of correction obtained by direct operation on the paretic superior oblique were gratifying. In some instances, it was necessary to perform supplementary recession of a secondarily contracted inferior oblique when the paresis was of long standing or a small recession of the opposite inferior rectus to

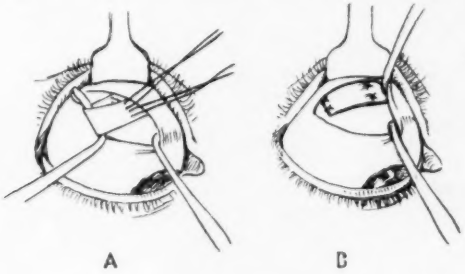


Fig. 2.—*A*, tucking of the superior oblique by temporal approach, without disturbing the superior rectus; *B*, tip of tuck anchored to sclera.

achieve final satisfactory results. No clearcut relation was found between the length of the tuck and the amount of correction obtained. It seems best to estimate the amount required by the appearance and feel of the muscle on the hook and then to adjust it to the proper tension. An estimate of this proper tension is gained by careful exposure and by testing of normal superior oblique muscles during routine enucleations.

Five cases are cited as examples of situations in which tucking of the superior oblique is useful. The other cases were similar.

ILLUSTRATIVE CASES

CASE 1.—*Congenital Paresis of Superior Oblique Muscles with Head Tilt, Vertical Diplopia and Asthenopic Symptoms.*

P. L., a 15 year old schoolgirl, had had head tilt to the right, headaches brought on by reading, occasional vertical diplopia and occasional deviation up and out of the left eye when very tired, as long as she could remember. Her mother stated

that for the first five years of the patient's life the left eye had turned up and in, until an anterior tenotomy of the left inferior oblique muscle had been performed. This first operation had resulted in some cosmetic improvement. On Nov. 25, 1944 she had 20/20— vision in each eye. The only abnormalities were in function of the extraocular muscles. The results of general physical examination and laboratory studies were normal. She customarily held her head tilted toward the right shoulder. There was definite lag in excursion of the left superior oblique (fig. 3). Muscle balance measurements, expressed in prism diopters, by cover test were [12]:

X 6 △
LH 18△

X 10△
LH 4 △

X 6 LH 12△
X 12 LH 16△

X 4 △
LH 45△

X 6△
LH Trace

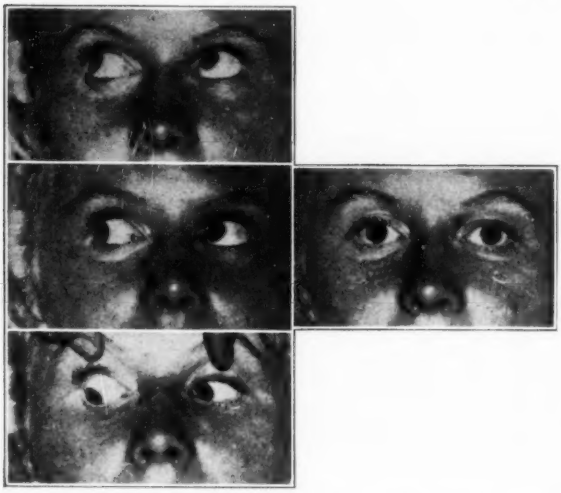


Fig. 3 (case 1).—Congenital paresis of left superior oblique (preoperative appearance).

With a red glass, diplopia could be elicited, increasing on the patient's looking to the right and down, with the left image lower. Fusion was good. A diagnosis of congenital paresis of the left superior oblique muscle was made.

On Sept. 6, 1945, with the patient under thiopental sodium (pentothal sodium®) anesthesia, a 12 mm. tuck in the left superior oblique muscle was performed by the third method described. The postoperative reaction was only moder-

12. In this tabulation, and in the accompanying tabulations, X and X′ indicate exophoria for distant and near vision; XT and XT′, exotropia for distant and near vision; S and ST and S′ and ST′, esophoria and esotropia for distant and near vision, respectively; RH and RH′, right hyperphoria for distant and near vision, and LH and LH′, left hyperphoria for distant and near vision.

ate, but an overcorrection persisted for nearly a month. At the end of this time satisfactory muscle balance was obtained and has been maintained ever since (fig. 4). All her symptoms have disappeared, and she has been able to enter college and study for long periods without discomfort, diplopia or head tilt. The last muscle balance measurements, made on Sept. 23, 1947, were:

X 2△ X 1△ LH 2△ X 4△
X 7△
X 2△ X 3△
LH 4△

CASE 2.—*Recurrent Amblyopia After Operation on Horizontal Rectus Muscles Overcome by Operation on Congenitally Paretic Superior Oblique.*

M. B., a 5 year old boy, had first been seen by an ophthalmologist in Baltimore for convergent strabismus and amblyopia ex anopsia of the right eye. After

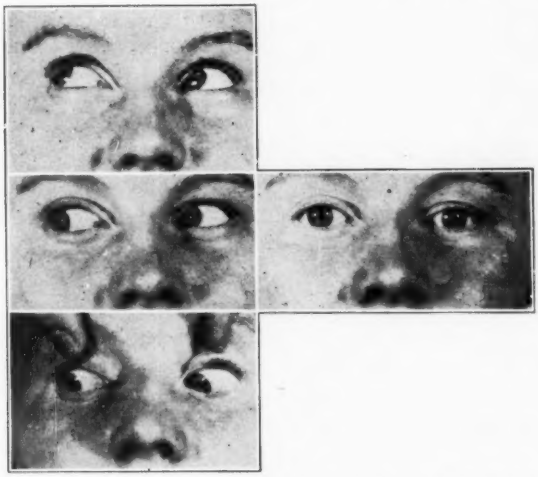


Fig. 4 (case 1).—Improvement in function of superior oblique after tucking.

correction of the amblyopia by training and occlusion, recession of the right medial rectus and resection of the right lateral rectus in 1940 had produced a good cosmetic result. Shortly thereafter his family moved to New York.

Examination on April 28, 1941 showed the corrected visual acuity to be 20/70 in the right eye, 20/20 in the left eye. There were operative scars over the horizontal rectus muscles of the right eye, but the eyes were otherwise structurally normal. Ocular excursions showed a lag in the right superior oblique (fig. 5). There was no pronounced head tilt and diplopia was not elicited. Muscle balance measurements by cover test were:

XT 10△ XT 5△ RH 4△ XT 7 △
ST' – XT'
XT 7 △ RH 12△
RH 4 △

Amblyopia was readily overcome by constant occlusion of the left eye. However, shortly after removal of the patch it recurred. Amblyopia was again overcome by occlusion and vertical prisms of 2 D., base down (right eye), and 2 D., base up (left eye), were prescribed. This correction also failed. On Dec. 5, 1941, with the patient under ether anesthesia, a 10 mm. tuck of the right superior oblique was performed by the first method. There were considerable postoperative reaction and an immediate overcorrection. These complications subsided in about six weeks, and normal muscle balance with normal vision in both eyes was maintained. Vision of 20/20 — in the right eye and 20/20 in the left eye has remained ever since.

The last muscle balance measurements, on June 29, 1945, were:

O O

RH ½△
X' 3△ RH' 1△

O O

Fig. 5 (case 2).—Congenital paresis of right superior oblique after horizontal surgical correction.

CASE 3.—*Intermittent Convergent Strabismus Corrected by Tucking of Congenitally Paretic Superior Oblique.*

S. S., an 8 year old boy, was brought in to the clinic by his mother on May 12, 1943. She complained that his eyes had turned in with increasing frequency since the age of 3 years. The use of "strong glasses" had helped at first, but at the time of consultation the eyes turned in most of the day, even with glasses. The only important event in his past history had been an appendectomy at the age of 7. The pediatric clinic reported that except for his eyes he was a normal child.

Examination of his eyes disclosed corrected vision of 20/30 + in each eye. He was wearing full atropine correction of a plus 5.00 sphere for the right eye and a plus 5.50 sphere for the left eye. With full correction there were

esophoria and right hyperphoria becoming a right hypertropia and right esotropia on removal of the glasses, and often with them on. On excursions there was obvious weakness of the right superior oblique muscle. When the eyes were straightened with glasses, the head tilted to the left. When fusion was broken, the head straightened. Muscle balance measurements were:

ST 14△ RH 5 △		ST 16△ RH 12△
	ST 12△ RH 9 △ ST' 18△ RH' 12△	
ST 20△		ST 20△ RH 26△

On Aug. 12, 1943, with the patient under ether anesthesia, a 10 mm. tuck of the right superior oblique was performed by the second method described. The postoperative reaction was moderate, and the immediate overcorrection soon subsided. From that time until Oct. 13, 1944, when the patient was last seen, function of the superior oblique was good, and the eyes were held straight without glasses at all times. Muscle balance measurements were:

S 6 △		S 8 △
	S 4 △ S' 12△ RH' 1△	
S 10△		S 10△ RH 4 △

Fig. 6 (case 2).—Function of paretic superior oblique muscle in both eyes restored by tucking.

CASE 4.—*Bilateral Paresis of Superior Oblique with Convergent Strabismus (Divergence Insufficiency).*

S. S., a 33 year old woman, came to the clinic complaining of "crossed eyes." In 1937 she had had a recession of her left medial rectus for the same complaint. Vision was 20/30 + in the right eye and 20/200 in the left eye. Except for a small scar over the left medial rectus, the eyes were structurally normal. There was obvious convergent strabismus with underaction of both superior oblique muscles. No head tilt or diplopia was noted. Amblyopia in the left eye could not be improved. Muscle balance measurements were:

ST 37△ RH-LH△		ST 30△ RH 6 △
	ST 56△ RH-LH ST' 40△ RH'-LH	
ST 40△ LH 12△		ST 50△ RH' 16△

A diagnosis of divergence insufficiency with bilateral paresis of the superior oblique was made. On Nov. 11, 1943, with the patient under general anesthesia,

the right lateral rectus was resected 10 mm. and the right superior oblique tucked 12 mm. by the first method. The left lateral rectus was resected 9 mm. and the left superior oblique tucked 10 mm. by the third method. There was definitely more reaction after operation in the right eye. After the reaction subsided, the eyes settled down to good position and excursions in all fields, with good depression by the superior oblique muscles (fig. 6). Muscle balance measurements on July 3, 1945 were:

XT Trace	XT 1△ RH 1△	XT Trace
	ST' 2△ RH' 3△	
LH Trace		RH 4△

CASE 5.—*Convergent Strabismus with Pareses of Superior Rectus and Superior Oblique in the Same Eye.*

M. K., a 5 year old girl, had had bilateral recession of the medial rectus for convergent strabismus at the age of 3. The eyes had appeared straight for about a year but then converged again. The refractive error was minimal. Vision

Fig. 7 (case 5).—Congenital pareses of right superior rectus and right superior oblique (preoperative appearance).

was 20/30 in each eye. Fixation was made with the right eye in all fields to the right, and with the left eye in all fields to the left. There were incomplete excursions in the field of the right superior rectus and the right superior oblique (fig. 7). Measurements by screening were:

ST 25△		ST 25△
LH10 △		RH 8 △
	ST 25△ RH 5△	
	ST' 28△ RH' 7△	
ST 24△		ST 28△
LH 8 △		RH 12△

In the field of the paretic right superior rectus, fixation was performed by the paretic eye with secondary deviation of the left inferior oblique. In the field of the paretic right superior oblique fixation was performed by the sound eye. Surgical intervention was planned accordingly; on May 8, 1944 both lateral rectus muscles were resected 9 mm., the left inferior oblique was receded 8 mm. and the right superior oblique was tucked 8 mm., by the third method. The post-

operative reaction was slight and the result satisfactory. Most recent measurements, on Oct. 13, 1945, were:

RH Trace		S 5△
	RH 4-LH 3△	
	RH' 2-LH' 3△	
O		O

PARESIS OF THE INFERIOR OBLIQUE

In like manner, the proper approach to a paretic inferior oblique muscle can be deduced. In this case, however, the problem of a muscle with important function in the reading position does not enter into the discussion. The principle of operating on the paretic muscle when the sound eye fixes in the field of the defect and weakening the secondarily deviating muscle when the paretic eye fixes in that field should be more strongly adhered to. Exception may have to be taken when the secondary deviation of a superior rectus is extreme and would require excessive recession of that muscle with resultant retraction of the upper lid. Such a situation is better faced by splitting the correction between strengthening the paretic inferior oblique and weakening the superior rectus of the opposite eye, even though all fixation is accomplished with the paretic eye. If too large a recession is performed on a superior rectus muscle, recession of the levator may later be required for cosmetic purposes. It also seems better, when such a divided surgical procedure is chosen, to perform the operation in two stages, with strengthening of the paretic inferior oblique as the first stage and with recession of the secondarily overacting superior rectus reserved to take up the balance. Isolated pareses are less common in the inferior oblique than in the superior oblique muscle, and these pareses are more apt to be found in complex combinations.

TECHNIC

The first approach tried was Wheeler's[1] advancement of the origin of the inferior oblique over the anterior rim of the orbit. Because results were erratic, a search was made for a more satisfactory technic. Because of the success with tucking of the superior oblique, tucking of the inferior oblique at its scleral insertion was next attempted.

This method was abandoned after 8 tucking operations because the bulk of the tuck tended to collide with, and adhere to, the overlying lateral rectus muscle. The next approach was resection at the insertion, after White.[13] This procedure seemed to obviate some of the disadvantages of the tucking but, in common with it, had an outstanding fault. Small corrections could be readily achieved, but large resections gave little or no further correction. The explanation of this strange phenomenon seems to be in the anatomy of the muscle itself. With a very short, practically negligible, tendon at its insertion the inferior oblique cannot be resected extensively without destroying enough of the contractile tissue of the muscle belly to defeat the purpose of the large resection. After 22 resections, it

13. White, J. W.: Paralysis of Superior Rectus and Inferior Oblique Muscle of Same Eye, Arch. Ophth. **27**:366 (Feb.) 1942.

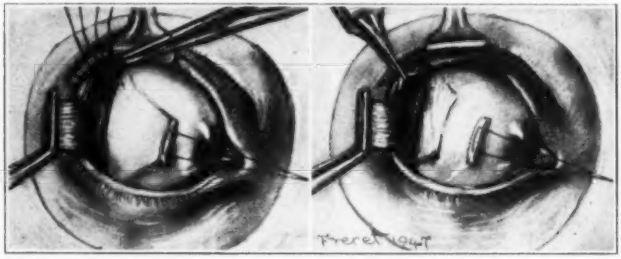
was determined that this operation must be reserved for cases requiring only small corrections.

The obvious procedure, then, seemed to be advancement of the insertion of the muscle. This was accomplished by temporarily detaching the lateral rectus, freeing the adhesions between the two muscle sheaths and advancing the inferior oblique along the sclera in its line of action (fig. 8*A*). When anatomic considerations precluded advancement to the degree desired, a small resection was added at the tip. Buried sutures of chromic surgical gut, silk or nylon were used to anchor the muscle to the sclera. It was found that a narrow-bladed hemostat was a more satisfactory muscle clamp in this situation than any of the conventional ones. Before the lateral rectus was replaced, a tongue of Tenon's capsule was brought down over the advanced inferior oblique to prevent any possible adhesions between the two muscles (fig. 8*B*).

This technic has been used in 15 advancements of 15 paretic inferior oblique muscles in 11 patients.

RESULTS AND ILLUSTRATIVE CASES

As in the case of tucking of the superior oblique muscle, no obvious relation was found between the number of millimeters of advancement



A B

Fig. 8.—*A*, advancement of inferior oblique under lateral rectus muscle; *B*, Tenon's capsule flap over advanced inferior oblique and under lateral rectus.

and the number of prism diopters gained. Three cases illustrating different situations in which this operation was useful follow.

Case 6.—*Congenital Paresis of the Inferior Oblique with Secondary Convergent Strabismus.*

J. D., an 11 year old boy, was referred on Aug. 8, 1945 for operation on a congenitally paretic inferior oblique muscle in the right eye. His mother stated that he was "born with a turned eye." On examination he was found to have marked weakness of the inferior oblique muscle of his right eye and convergent strabismus. Vision was 20/20 in each eye, and fixation alternated. When he fixed with the right (paretic) eye, a marked vertical deviation developed. When he fixed with the left (nonparetic) eye, there was a pronounced right convergence. Except for the motor anomaly (fig. 9) the eyes were normal. Measurements by screening gave the following values:

ST 55△		ST 60△ LH 45△
	ST 50△ LH 18△ ST′ 60△ LH′ 18△	
ST 60△ RH-LH		ST 60△ LH 20△

On Sept. 27, 1945, with the patient under ether anesthesia, 5 mm. was resected from the tip of the right inferior oblique, and the new end was advanced 11 mm. along the sclera and a Tenon's capsule apron drawn over this muscle, as shown in figure 8. A resection of 8 mm. was done on the right lateral rectus as it was replaced. This surgical procedure on the lateral muscle was not expected to be adequate, but the rest of the correction was reserved for a second stage.

The postoperative course was uneventful, leaving some undercorrection both vertically and horizontally, as follows:

ST 40△		ST 40△
RH 4 △		LH 18△
	ST 40△ LH 14△	
	ST' 45△ LH' 18△	
ST 40△		ST 40△
RH Trace		LH 10△

On Feb. 21, 1946 the second operation was performed, with the patient under ether anesthesia. Recessions of 2.5 mm. were performed on both medial rectus muscles and recession of 4 mm. was done on the left superior rectus muscle. This procedure resulted in a fairly satisfactory result, with good, but not

Fig. 9 (case 6).—Congenital paresis of right inferior oblique (preoperative appearance).

perfect, elevation in the field of the right inferior oblique (fig. 10). The most recent muscle measurements, on Dec. 17, 1947, were:

RH 2△		LH 8△
	S 8△ LH 3△	
	S' 8△ LH' Trace	
RH 3△		LH 4△

No further surgical correction is indicated.

CASE 7.—*Paresis of Both Inferior Oblique Muscles in Convergent Strabismus.*

B. B., 6 years old, a surgeon's daughter, was brought in by her father, who stated that her eyes had been "out of line" both horizontally and vertically all her life. She was otherwise a normal, healthy girl. Examination disclosed vision of 20/20 in each eye and globes that were normal except for the motor function. There was obvious underaction of the lateral rectus and the inferior oblique muscles of both eyes.

Measurements of muscle balance were:

ST 40△		ST 47△
RH 20△		LH 16△
	ST 40△ LH 6△	
	ST' 30△ RH' 4△	
ST 30△		ST 28△
RH 12△		LH 7 △

On Dec. 6, 1945, with the patient under ether anesthesia, the inferior oblique muscles were advanced (10 mm. for the right eye and 12 mm. for the left eye),

and the lateral rectus muscles were resected (8 mm. for the right eye and 10 mm. for the left eye). After a mild postoperative reaction, the eyes settled down to normal function and position. The most recent measurements, on Oct. 21, 1947, showed a trace of exophoria in the primary position and orthophoria in the fields (fig. 11).

CASE 8.—*Paresis of the Inferior Oblique as Part of "Double Elevator Palsy."*

J. M., a 7 year old girl, was referred from another hospital for surgical treatment of "double elevator palsy." The parents complained that the eyes had "never focused" and that most of the time one was closed.

Fig. 10 (case 6).—Function of right inferior oblique improved by advancement of the insertion.

Fig. 11 (case 7).—Function of both inferior oblique muscles restored by advancement.

On examination she was found to have pseudoptosis and partial amblyopia (20/70) in the right eye. The left eye had normal (20/20) vision. When she fixed with the left eye, the right lid drooped to occlude vision in the slightly depressed and adducted right eye (lowest photograph, fig. 12). When she fixed with the right eye, the lid opened and the left eye turned up and in (center

photograph, fig. 12). On attempts to look up, the right eye failed in all fields (upper photographs, fig. 12). Measurements of muscle balance were:

LH 80△ LH 65△
ST 50△ ST 50△

LH 50△ (right fixation)
LH 30△ (left fixation)
ST 50△

LH 40△ LH 40△
ST 60△ ST 55△

She had obvious paresis of both elevator muscles of the right eye with secondary pseudoptosis and convergent strabismus. Since fixation was made with the left eye in all fields, the theoretically proper approach would be to the right superior rectus and right inferior oblique muscles. However, it was felt that adequate correction could not be achieved with these muscles alone, and the left elevator muscles should also be weakened. On March 28, 1946, with the patient under ether anesthesia, the right inferior oblique was resected 4 mm. and the tip advanced

Fig. 12. (case 8).—Double palsy of the elevator muscles (preoperative appearance).

14 mm., the right superior rectus resected 6 mm., the right lateral rectus resected 10 mm., the left inferior oblique receded 9 mm. and the left superior rectus receded 3 mm., in one operation. The immediate postoperative reaction was moderately severe. On the fifth postoperative day measles developed, and the course of convalescence was stormy. By May 3 (two months after operation) the eyes were quiet and well healed, with considerable improvement. Measurements of muscle balance at that time were:

LH 9 △ LH 8△
ST 12△

ST 5 △ LH 3△
ST' 12△ LH' 3△

LH Trace LH Trace
ST 8△

Some pseudoptosis remained. The child disappeared from observation after that time, and all attempts at follow-up have been unsuccessful. Final measurements and postoperative photographs have not been obtained.

COMMENT

The technics described in a previous section, and illustrated in figures 2 and 8 have proved highly satisfactory in direct surgical strengthening of paretic oblique muscles. Certain factors which experience with these operations has shown deserve comment.

These methods, although they are technically somewhat more difficult than the commoner operations of the rectus muscles, are entirely practicable. If they are performed neatly and without unnecessary trauma to tissues, they do not cause much more reaction than do the conventional muscle operations. They therefore add to our surgical armamentarium procedures which make it possible to plan and effect

TABLE 1.—*Correction Obtained by Tucking the Superior Oblique Muscle*

Tuck, Mm.	Correction Obtained					
	Distant Vision		Near Vision		Field	
	△	△/Mm.	△	△/Mm.	△	△/Mm.
12	12	1.0	14	1.2	41	3.4
10	3	0.3	4	0.4	10	1.0
10	9	0.9	11	1.1	22	2.2
12	..	...	..	...	12	1.0
10	1	0.1	3	0.3	12	1.2
8	4	0.5	5	0.6	12	1.5
12	10	0.8	10	0.8	18	1.5
10	7	0.7	7	0.7	10	1.0
12	10	0.8	12	1.0	21	1.8
10	14	1.4	14	1.4	21	2.1
10	6	0.6	6	0.6	19	1.9
16	36	2.3	..	...	32	2.0
14	12	0.9	12	0.9	38	2.7
8	2	0.3	3	0.4	14	1.8
15	25	1.7	26	1.7	..	...
20	26	1.3	30	1.5	..	...
18	10	0.6	14	0.8	40	2.2
8	2	0.3	4	0.5	12	1.5

correction in the more complicated muscle cases according to the best accepted principles. Undesirable compromises are less often necessary, and better functional results can be obtained.

The greatest difficulty in these operations is the necessity of judging the amount of correction to be made by the appearance and feel of the muscle at operation. Perhaps this difficulty is to be expected, for it is well known that exact surgical correction of even the simple horizontal deviations cannot be exactly calculated in advance but must be modified after the muscle is exposed. In spite of this difficulty, results well within acceptable limits [14] can be obtained. Analysis of the number of prism diopters gained in the primary position and in the field of

14. Dunnington, J. H., and Wheeler, M. C.: Tr. Am. Acad. Ophth. **46**:206, 1941.

action of the muscle for each millimeter of surgical correction are shown in table 1 for the superior oblique muscle and in table 2 for the inferior oblique muscle. It can be seen that variation is wide both in tucking of the superior oblique and in advancement of the inferior oblique. An average of the results obtained yields a figure of nearly 1 Δ gain per millimeter of muscle correction, and to be expected in the primary position of nearly 2 Δ in the field of action of the muscle. However, the individual variations are wide.

The figures are practically the same for tucking of the superior oblique and advancement of the inferior oblique muscle. There seems to be a correlation between the appearance of the muscle or tendon and the amount required, but there is no way to express this mathematically. There appears to be no way by which to judge the amount

TABLE 2.—*Results of Advancement* of the Inferior Oblique*

Advancement, Mm.	Correction Obtained					
	Distant Vision		Near Vision		Field	
	Δ	Δ/Mm.	Δ	Δ/Mm.	Δ	Δ/Mm.
16	4	0.3	0	0	27	1.7
10	6	0.6	..	...	16	1.6
12	..	...	4	0.3	20	1.6
14	13	0.9	18	1.3	47	3.4
10	2	0.2	1	0.1	4	0.4
13	10	0.8	12	0.9	27	2.1
11	16	1.5	16	1.5	24	2.2

* The other inferior oblique operations were sufficiently complicated by additional surgical correction of the vertical muscles that the exact amount attributable to individual operations on the oblique muscles could not be accurately estimated.

of surgical correction required better than that of adjusting the tension according to the surgeon's sense of touch.

A word should be added about immediate overcorrection. In all satisfactory cases the appearance in the immediate postoperative period is one of pronounced overcorrection. If this is not present, the final result may be expected to be an undercorrection.

SUMMARY

The reasons for direct operative procedures to strengthen a weak oblique muscle are cited. Technics for tucking a paretic superior oblique and advancing a paretic inferior oblique muscle are described. Illustrative cases in which the three technics were successfully applied are reported. The methods of judging the amount of operation to be performed are discussed.

525 East Sixty-Eighth Street.

Ophthalmologic Reviews

AQUEOUS VEINS AND THEIR SIGNIFICANCE FOR PATHOGENESIS OF GLAUCOMA

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CINCINNATI

DETECTION AND QUALITIES OF THE AQUEOUS VEINS

ALL OVER the body, fluid may leave or enter capillaries according to the relative values of tissue pressure and intravascular pressure and osmotic and colloidal osmotic pressure which prevail inside and outside the vessel walls. An individual capillary may yield tissue fluid in its proximal part and accept tissue fluid in its distal part.[1] In the human eye, there is superimposed on this universal water exchange through the capillary endothelium a continuous gross circulation of fluid, with its source in the ciliary body and its goal in the chamber angle.[2] From the angle of the anterior chamber fluid seeps through the trabecular meshwork into the canal of Schlemm, hence through the scleral venous meshwork and, finally, into conjunctival and episcleral veins, where it may become biomicroscopically visible.[3] This circulation of fluid was postulated by Leber [2a] and his followers [4] but was never definitely proved or really observed. For decades, many investigators denied the existence of a continuous production and elimination of fluid into and out of the eye.[5] The detection of the aqueous veins, in 1941,

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The Snyder Ophthalmological Foundation made grants in aid for research on aqueous veins.

1. Best, C., and Taylor, N.: Physiological Basis of Medical Practice, ed. 4, Baltimore, Williams & Wilkins Company, 1945, pp. 23-26.

2. (*a*) Leber, T.: Die Zirkulation und Ernährungsverhältnisse des Auges, in Graefe, A., and Saemisch, T.: Handbuch der gesammten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1903, vol. 2. (*b*) Duke-Elder, W. S., and Davson, H.: The Present Position of the Problem of the Intraocular Fluid and Pressure, Brit. J. Ophth. **32**:555-569 (Sept.) 1948.

3. Ascher, K. W.: Aqueous Veins: Preliminary Note, Am. J. Ophth. **25**: 31-38 (Jan.) 1942.

4. (*a*) Seidel, E.: Mikroskopische Beobachtungen über den Mechanismus des Abflusses aus der Vorderkammer des lebenden Tieres, Arch. f. Ophth. **112**:170 (March) 1923. (*b*) Serr, H.: Mechanik der Augendruckschwankungen beim primären Glaukom, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **45**:225, 1925.

5. Hamburger, C.: Antwort auf Seidels Aufsatz, zum Beweis der Filtrations Theorie, Klin. Monatsbl. f. Augenh. **72**:206, 1924. Weiss, O.: Physiologie der

(Footnote continued on next page)

disproved the assumption of stagnation of the intraocular fluid. With a continuous flow of intraocular fluid leaving the canal of Schlemm through the aqueous veins, a continuous production of aqueous humor is postulated.[6]

Aqueous veins are biomicroscopically visible channels resembling blood vessels and connecting the canal of Schlemm and its outlets into the deep scleral meshwork with the superficially visible conjunctival and episcleral vessels. They can be found in almost every normal human eye[7]; in eyes of experimental animals[8] they can be photographed, although not without difficulty,[9] and repeated photographs have proved that they are stable anatomic features which do not change their location and course after months and years.[10] Larger aqueous veins may be detected by means of a simple magnifying loupe of 10 diameters.[11]

It may seem incredible that these important structures escaped the attention of physiologists and ophthalmologists for decades after the introduction of slit lamp microscopy. This was due to several factors: lack of interest in studying the limbal region of normal eyes; the small size of many of these aqueous veins when they arise in the limbal meshwork; the blood which often joins the clear fluid while streaming through the deep scleral meshwork and after leaving the sclera, and the traditional, but disadvantageous, custom of using only the narrow slit of the corneal microscope. According to a drawing published in 1934, Graves[12] probably saw an aqueous vein, and so possibly did

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Ernährung und der Zirkulation des Auges, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1932, vol. 2. Magitot, A.: Le liquide endoculaire et la nutrition du globe, in Bailliart, P.; Coutela, C.; Douglas, N., and Bucy, P. C.: Traité d'ophtalmologie, Paris, Masson & Cie, 1938, vol. 2. Fischer, F. P.: Der Wasserhaushalt des Auges und seiner Teile, Docum. ophth. **1**:79-160, 1938.

6. Ascher, K. W.: Physiologic Importance of the Visible Elimination of Intraocular Fluid, Am. J. Ophth. **25**:1174-1209 (Oct.) 1942.

7. de Vries, S.: De zichtbare afvoer van het kamerwater, Amsterdam, Drukkerij Kinsbergen, 1947.

8. Schmerl, E.: Significance of Action of Paredrine on the Ocular Tension of Rabbits, Am. J. Ophth. **30**:186-190 (Feb.) 1947.

9. (*a*) de Vries.[7] (*b*) Gartner, S.: Blood Vessels of the Conjunctiva, Arch. Ophth. **32**:464-476 (Dec.) 1944.

10. Ascher, K. W.: Search for Aqueous Veins, Am. J. Ophth. **31**:105-106 (Jan.) 1948.

11. Ascher, K. W.: Further Observations on Aqueous Veins, Am. J. Ophth. **29**:1373-1387 (Nov.) 1946.

12. Graves B.: Certain Clinical Features of the Normal Limbus. Brit. J. Ophth. **18**:305-368 (June) 1934.

others before and after him, without paying due attention to these features. Broad aqueous veins, arising from a scleral emissary, are more easily visible; but most of them are much smaller, and, according to de Vries,[7] the search for them can be compared to the search for the needle in the haystack.

From Theobald's [13] diagrams of the outlets of Schlemm canals, it is evident that the widest outlets were located near the horizontal meridian of all 3 eyes investigated; this region corresponded to the origin of the majority of the aqueous veins.[6] Arising from the limbal meshwork as narrow capillary vessels, or from the depths of the scleral tissue with a slightly wider figure 6–like or U arch, or as wide channels from a scleral emissary, aqueous veins take a rather straight course through the conjunctival or the subconjunctival tissue. They are to be recognized by their pale or limpid coloration, which may vary during the observation and, in many instances, by a layering, consisting of two, three or more strata of differently shaded fluids, the layers running parallel to each other and to the wall of the vessel. The length of an aqueous vein varies from a fraction of a millimeter to 1 cm. or slightly more; what appears to be an unusually long aqueous vein is often a recipient vessel. The width of aqueous veins is between 0.01 and 0.1 mm. Below the point of their emptying into the regular conjunctival or episcleral veins, their clear contents are visible within the recipient vessel for a variable distance, as when a clear creek empties into a muddy river (fig. 1). If a large aqueous vein is found in one eye, a similar one may be detected in the fellow eye symmetrically located.[14] The eyes of parents and children may show aqueous veins at similar locations and with a similar course and qualities.[6]

Stratified currents are not a unique finding; they may be observed in various other organs, as in the tongue and the web of the frog,[15] in the mesentery and the spleen of the mouse [16] and occasionally inside the human eye of the canal of Schlemm when viewed gonioscopically.[17] The same picture may be due to a partition of the canal, however.

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13. Dvorak-Theobald, G.: Schlemm's Canal: Its Anastomoses and Anatomic Relations, Tr. Am. Ophth. Soc. **32**:593, 1934; unpublished recent data.

14. Ascher,[6] de Vries.[7]

15. Thoma, R.: Experimentell-mathematische Behandlung des Blutkreislaufs, in Abderhalden, E.: Handbuch der biologischen Arbeitsmethoden, Berlin, Urban & Schwarzenberg, 1927, vol. 2, pt. 5, sec. 4, chap. 2, p. 1118, Jaeger, A.: Anordnung und Stellung der rothen Blutkörperchen im strömenden Blut, Arch. f. d. ges. Physiol. **235**:715, 1935; Arch. f. Augenh. **110**:148, 1937.

16. Kniseley, M. H.: Microscopic Observations of the Circulatory System of Living Unstimulated Mammalian Spleens, Anat. Rec. **65**:25 (April) 1936.

17. Troncoso, M. U.: Gonioscopy and Its Clinical Applications, Am. J. Ophth. **8**:433-449 (June) 1925.

Embryologically, anatomically and physiologically, the canal of Schlemm, the scleral meshwork and the aqueous veins form a biologic unit charged with the elimination of intraocular fluid.[6] According to computations by various authors, an average of 5 cu. mm. of fluid is eliminated each minute from the anterior chamber via the canal of Schlemm [18]; considering the diameters of the aqueous veins and the speed of the fluid passing these channels, one may arrive at similar figures for the amount of fluid leaving the eye through the aqueous veins.[14] Goldmann [19] reported on experiments which he performed to prove that the contents of the aqueous veins is really aqueous humor.

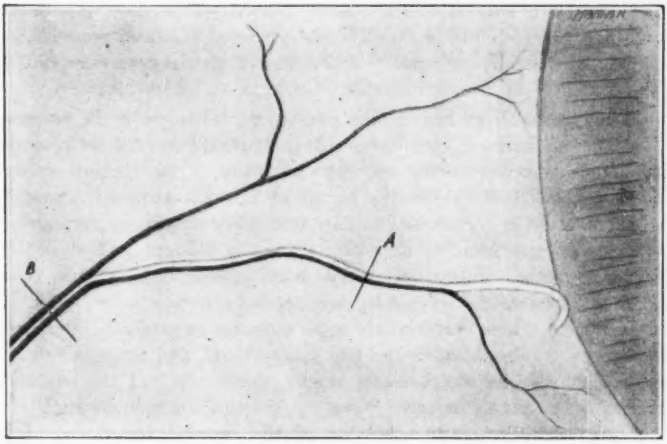


Fig. 1.—Origin and course of a typical aqueous vein (left eye of a white man aged, 48). An aqueous vein starts from the lower nasal portion of the limbus with an arclike origin and empties into a regular conjunctival vein. Below the junction, two strata are visible (*A*), the clear one being the aqueous section of the recipient vessel. After joining another vein (*B*), the recipient vessel contains two peripheral red layers and one central aqueous stream (Ascher,[3] page 33).

Physiologic and physical factors, such as heart action, respiration and illumination, have a characteristic influence on the contents of the aqueous veins; illumination enhances the elimination of fluid in many, but not in all, these veins, while pressure of the lids interferes with it in practically every aqueous vein.[6]

18. Duke-Elder, W. S.: Text-Book of Ophthalmology, London, Harry Kimpton, 1932, vol. 1, p. 456. Kinsey, V. E.; Grant, W. M., and Cogan, D. G.: Water Movement in the Eye, Arch. Ophth. **27**:242-252 (Feb.) 1942.

19. Goldmann, H.: Weitere Mitteilung über den Abfluss des Kammerwassers, Ophthalmologica **112**:344-349 (Dec.) 1946.

Epinephrine reduces, and ethyl morphine hydrochloride increases, the amount of blood in the aqueous veins and in their recipient vessels [20]; homatropine interferes with the elimination of fluid, while physostigmine and pilocarpine increase the output of clear fluid.[21]

AQUEOUS VEINS IN GLAUCOMATOUS EYES

In glaucomatous eyes an increase in clear fluid will appear in the aqueous veins after instillation of pilocarpine.[22] This important fact was photographically recorded by Gartner [9b] in 1944. In congested eyes the aqueous veins become invisible, but they reappear when the congestion subsides; in glaucomatous eyes they may become visible after successful surgical intervention.[22] Periods of increased intraocular pressure coincide with, or may even be preceded by, an increased blood content of the aqueous veins.[23] These minute changes were studied by Thomassen [23a] at hourly intervals.

Sufficient evidence has accumulated since 1941 to prove the presence of a minutely balanced and changeable pressure differential between the two fluids, aqueous humor and blood, flowing in the recipient vessel of an aqueous vein. When, by means of a small cotton applicator, a recipient vessel is compressed distally from the emptying of an aqueous vein, either expulsion of all blood from the blocked section of the vessel or filling with red blood cells may be observed.[24] When compression is released, the picture reappears as it was prior to either phenomenon. These reactions are apparently due to pressure differences in the sources from which the two fluids, blood and aqueous humor, derive, namely, the deep scleral venous meshwork and the canal of Schlemm. For purely descriptive reasons, the aqueous influx was called [3] the glass rod phenomenon because of the resemblance of the clear

20. (*a*) Ascher, K. W.: Local Pharmacologic Effects on Aqueous Veins, Am. J. Ophth. **25**:1301-1309 (Oct.) 1942. (*b*) Goldmann, H.: Abfluss des Kammerwassers beim Menschen, Ophthalmologica **11**:146-152 (Feb.-March) 1946. (*c*) de Vries.[7]

21. Ascher.[20a] Gartner.[9b]

22. Ascher, K. W.: Glaucoma and Aqueous Veins, Am. J. Ophth. **25**:1309-1315 (Oct.) 1942.

23. Ascher, K. W.: Backflow Phenomena in Aqueous Veins of Normal and of Glaucomatous Eyes, Am. J. Ophth. **27**:1074-1089 (Oct.) 1944.

23a. Thomassen, T. L.: On Aqueous Veins, Acta ophth. **25**:369-376 (Dec.) 1947.

24. (*a*) Ascher (footnotes 3, 6, 23 and 11) (*b*) de Vries.[7] (*c*) Ascher, K. W., and Spurgeon, W. M.: Compression Tests on Aqueous Veins of Glaucomatous Eyes: Application of Hydrodynamic Principles to the Problem of Intraocular Fluid Elimination, Am. J. Ophth. **32**: Part II: pp. 239-251, 1949. (*d*) Goldmann, H.: Ueber Abflussdruck und Glasstabphänomen, Ophthalmologica **116**:195-198 (Oct.-Nov.) 1948.

vessel to a rod of glass; a more unequivocal name, aqueous influx phenomenon,[24c] will be used in the future; the opposite effect will be called the blood influx phenomenon, instead of the original term "negative glass rod phenomenon." Neither represents an incidental or transient quality; whenever the test is repeated, the same response will be found, even after months and years, and is characteristic of the particular vessel. De Vries [7] was the first to make photographic records of the blood influx phenomenon. He made the very important observation that the blood influx phenomenon was found in aqueous veins with low speed of flow, e. g., 1.5 mm. per second, while aqueous veins with a measured velocity of 3.5 or 7.5 mm. per second showed the aqueous influx phenomenon.

One might expect that the high pressure prevailing in the anterior chamber of the glaucomatous eye would be transmitted to the canal of Schlemm and hence into its invisible outlets, as well as into the visible aqueous veins; that therefore the aqueous outflow might be more vigorous in glaucomatous eyes, and that therefore the aqueous influx phenomenon might occur in glaucomatous eyes oftener than in eyes with normal pressure. A similar assumption made Verhoeff [25] ask whether in eyes with wide angle glaucoma the clear stream in the aqueous veins might be longer and wider than in eyes with normal pressure. Neither is this so, nor is the aqueous influx phenomenon the rule in the aqueous veins of glaucomatous eyes.[26] The compression of the recipient vessels in such eyes is almost always followed by blood influx, whereby the venous blood streams in a direction opposite its normal flow, namely, not from but back toward the limbus. One should expect, therefore, that on gonioscopic observation blood would be seen rushing into the canal of Schlemm, as it often does in eyes with normal intraocular pressure. Bangerter and Goldmann,[27] as well as Kronfeld and associates,[28] found, however, that backflow of blood into the canal of glaucomatous eyes is observed only as an exception. This contradictory behavior—backflow of blood toward the canal and failure of blood to enter the canal—can be explained by the assumption of narrowing of the outlets of the canal in glaucomatous eyes.[29] Sclerosis of the trabecular meshwork would explain only the influx of blood into the

25. Verhoeff, F. H., in discussion on Ascher,[20a] p. 1314.

26. Ascher.[22] de Vries.[7] Goldmann.[24g]

27. Bangerter, A., and Goldmann, H.: Gonioscopy in Primary Glaucoma and Choice of Operation, Ophthalmologica **102**:321-350 (Dec.) 1941.

28. Kronfeld, P. E.: McGarry, H. T., and Smith, H. E.: Gonioscopic Study on the Canal of Schlemm, Am. J. Ophth. **25**:1163-1173 (Oct.) 1942.

29. Ascher (footnotes 11 and 23). Ascher and Spurgeon.[24f]

blocked recipient vessel and into the aqueous vein, but with a low pressure inside the canal there would be no reason that blood should not stream back into it, as it does in eyes with normal pressure.

Another series of observations gave confirmation of the assumption that at least in some glaucomatous eyes the outlets of the canal of Schlemm are narrower than in eyes with normal pressure: pressure applied to the eyeball externally, and far from any visible aqueous vein, may increase the clear stream in aqueous veins and in recipient vessels.[6] This effect of increase in pressure, which I observed prior to 1942, was carefully studied by Goldmann [30] in Berne and by de Vries,[7] in Amsterdam, in 1946. Using a spring balance, Goldmann measured the amount of external pressure which was needed to widen the diameter of the clear stream in a recipient vessel. Whereas in normal eyes an average of 8 Gm. of external pressure sufficed for this effect, in glaucomatous eyes decidedly higher values, up to 30 Gm. and more, were necessary. In eyes with normal pressure, however, Goldmann found instances in which similarly high values of pressure increment were required in order that a perceptible increase in the clear contents of the recipient vessels should appear. These values were discarded by Goldmann because of their unusually high range. They were noted in eyes with normal intraocular pressure, in the case both of narrow recipient vessels and of those with an unusually slow current. Thus, the high readings are characteristic not of glaucoma but of narrowness of the particular vessel or, most probably, of its origin deep in the scleral meshwork, as suggested by the slow current. In other words, for eyes with normal pressure both high and low increments in pressure are found, whereas for glaucomatous eyes only high readings are present so long as the ocular pressure is elevated.

Mathieu and Weekers [31] encountered aqueous veins less frequently in eyes subjected to iridencleisis than in normal eyes and in eyes with other types of untreated and treated glaucoma. They found aqueous veins in 49 of 50 eyes with normal pressure; a similar ratio was obtained for eyes observed after cataract operation, for eyes with untreated glaucoma and for eyes the pressure of which had been reduced by miotics. Ten eyes subjected to cyclodiathermy several months prior to the investigation showed at least one aqueous vein each. Investigations performed several months, or even years, after iridencleisis showed a ratio of 19 eyes with aqueous veins to 14 eyes without aqueous veins.

30. Goldmann, H.: Studien über den Abflussdruck des Kammerwassers beim Menschen, Ophthalmologica **114**:81-94 (Aug.) 1947.

31. Mathieu, P., and Weekers, R.: Contribution à l'étude des veines aqueuses, Bull. Soc. belge d'opht., November 1948.

In order to study a larger series of eyes with complete recovery after iridencleisis, Weekers and Weekers [32] pooled their own cases and those observed by François [33] and found, among 106 eyes, 56 with and 46 without aqueous veins, the findings for 4 eyes being listed as questionable.

In discussing the possible causes of the rare occurrence of aqueous veins after iridencleisis, Mathieu and Weekers excluded (1) postoperative hyperemia, because this had subsided long before the examination; (2) scar formation, which could not possibly be responsible for absence of aqueous veins along the whole limbal circumference, and (3) a peculiar thickening and milky appearance of the conjunctiva, which occurred in very few cases. The authors found, however, that the aqueous veins were less frequent in the series of hypotonic eyes (in 18 of 50) than in the series with pressure readings higher than 15 mm. of mercury; in the latter 38 of 56 eyes showed aqueous veins. They concluded than in an eye with hypotony due to iridencleisis the surgically formed channel might carry the bulk of the fluid which leaves such an eye.

First Histopathologic Confirmation.—Unintentionally, Focosi [34] offered what might be the first histopathologic confirmation of my assumption that narrowing or obstruction of the canal outlets are connected with, if not the cause of, a certain type of glaucoma. Focosi described the case of a man aged 44 who, a few hours after operation for glaucoma in his right eye, had his first glaucomatous attack in his formerly normal (left) eye. This left eye had, prior to the operation on the other eye, a shallow chamber but normal intraocular pressure, a normal papilla of the optic nerve, normal visual acuity and a normal visual field. The glaucomatous attack of the intact eye was controlled by miotics before nightfall. Only a few deep corneal folds were visible during the succeeding days. Eight days after the operation, hemoptysis ended the patient's life. The left globe was embedded in pyroxylin, and histologic examination revealed partial occlusion of the chamber angle. Corresponding to the sealed chamber angle, a part of Schlemm's canal was obliterated, and the uncollapsed part of the canal, for a short distance, was obstructed by an amorphous, probably calcareous, cast. In this area one of the canal outlets contained a similar deposit, which stained deeply with hematoxylin and was granular in appearance. Since

32. Weekers, L., and Weekers, R.: Les fondaments physiopathologiques de l'iridenclesis, Ophthalmologica, to be published.

33. François, J.: La gonioscopie, Louvain, Librarie rue Fonteyn, 1948.

34. Focosi, M.: Osservazioni istologiche su di un caso recente di glaucoma acuto, Boll. d' ocul. **27**:209-227 (June) 1948.

only one of the outlets was obstructed, Focosi justly refrained from overestimating the significance of this first report of a glaucomatous eye with histologically verified occlusion of an outlet of Schlemm's canal. According to Maggiore [35] and Theobald,[13] there are between twenty and thirty canal outlets in every human eye; the smallest outlets have diameters measuring 5 and 30 microns; the diameters of the largest outlets, however, are 50 and 165 microns. It is reasonable to assume that an outlet with a cross sectional area sixty times as large as a small cross sectional area will be more effective for elimination of fluid. In some eyes, only a few large outlets may carry the main bulk of the

Fig. 2.—Obstruction of canal outlet; histologic confirmation (eye of a man aged 44, who died eight days after his first short glaucomatous attack). The arrow points toward the calcareous cast in the canal outlet (Focosi [34]).

intraocular fluid; occasionally, it may be even one large outlet, as suggested by certain unusually wide aqueous veins, shown in de Vries's photograph no. 33a.[7] In eyes of this type, occlusion of a few, or even of one, outlet may result in marked increase in intraocular pressure. By application of hydrodynamic principles to the problems of elimination of intraocular fluid, these effects can be formulated mathematically.[24c] In Focosi's specimen, the closure of the chamber angle and the collapse of the walls of the canal may have been of greater pathognomonic sig-

35. Maggiore, L.: Struttura, comportamento e significato del canale di Schlemm, Ann. di oftal. **40**:317-462, 1917.

nificance than the obstruction of one outlet by the calcareous cast; nevertheless, Focosi's observation showed the trend of further research and proved that the expected occlusion of canal outlets may be verified histologically.

APPLICATION OF HYDRODYNAMIC PRINCIPLES TO PROBLEMS OF ELIMINATION OF INTRAOCULAR FLUID

Mathematical evaluation by Spurgeon [24c] proved the significance of the diameter of the outlet for the pressure in the outlets and inside the canal of Schlemm. Reduction in the diameter of an outlet from 30 to 20 microns may raise the pressure inside the canal from 16 to 38 mm. of mercury. According to Poiseuille's equation, in which the diameter of the outlet is expressed in the fourth power, a slight reduction in the diameter will be followed by a considerable increase in the pressure at the origin of the outlet.

In addition to a narrowing of the outlets, reduction in their number will, to a mathematically calculable extent, influence the pressure inside the canal of Schlemm. If one half of all outlets are obliterated, say fifteen of thirty, the pressure in the canal, according to Poiseuille's equation, will be raised from 20 to 30 mm. of mercury. Such a reduction in the number of outlets may be expected to occur after a cataract incision unless it is assumed that the severed ends of the outlets can find each other again. Exact suturing of the lips of the wound in proper apposition will increase the chances of reapproximation of the cut ends of the canal outlets—another indication for exact suturing of the cataract incision. Severing of the larger outlets, which are found chiefly near the horizontal meridian, will endanger the eye more than that of smaller outlets.

I shall not enter into mathematical details, but the fact should be mentioned that most of the canal outlets are not circular but elliptic in cross section [35a] and that, according to the modified Poiseuille equation, a greater increase in pressure is to be expected for elliptic cross sections than for circular cross sections of the same square area.[36]

CONCLUSION AND SUMMARY

The elimination of intraocular fluid can be studied biomicroscopically.

Considered as a unit of clinicopathologic importance, the canal of Schlemm and the aqueous veins show parallel changes in congestion and in inflammation; in primary simple glaucoma, their responses are apparently contradictory so far as blood has a tendency to overwhelm the aqueous humor and to stream back against, but not into, the canal, which

35a. Dvorak-Therbald.[33] Maggiore.[35]

36. Milne-Thomson, L. M.: Theoretical Hydrodynamics, London, The Macmillan Company, 1938, pp. 517-519.

in eyes with normal pressure often becomes filled with blood during gonioscopic observation. These apparent contradictions can be explained in at least some cases of glaucoma by the assumption of a narrowing of the canal outlets.

TABLE 1.—*A Unit of Clinical Significance: The Canal of Schlemm, Its Outlets and the Aqueous Veins*

They Show During	Gonioscopic Appearance of the Canal	Biomicroscopic Appearance of the Aqueous Veins
Inflammation and congestion	Blood often enters the canal	Aqueous veins filled with blood; therefore not discernible
Primary simple glaucoma	Blood rarely enters the canal	After compression of the recipient vessel, blood enters the aqueous vein: blood influx phenomenon
Congestive glaucoma	Canal rarely visible	Aqueous veins carry blood from posterior uvea
Glaucoma controlled by miotics or operation	Blood may enter the canal	Aqueous veins reappear; clear fluid may expel blood from blocked recipient vessel: aqueous influx phenomenon

TABLE 2.—*Effect of Width of Canal Outlets and of Aqueous Veins*

Effect on:	Wide Outlets	Narrow Outlets
Gonioscopic Findings		
Backflow of blood into the canal of Schlemm.....	Frequent	Rare
Biomicroscopic Findings		
(*a*) Rate of flow of aqueous humor, observed on recipient vessel	High	Low
(*b*) Compression test, performed on recipient vessel, observed on aqueous vein: influx phenomenon	Aqueous influx	Blood influx
(*c*) Increment pressure test, performed on cornea, observed on recipient vessel: dynamometric readings	Low	High
Intraocular Pressure		
Tonometric readings.....	Normal if no other pathology	High if many outlets are narrow

No claim is made that this hypothesis can explain all cases of primary simple glaucoma; it is proposed in order to stimulate further research—clinical, histologic and experimental. A large number of new facts have accumulated, and there is, of course, ample space for interpretation; the evidence afforded by gonioscopy and by biomicroscopy is far from complete, and much remains to be done in order that one may approach the problem of glaucoma more satisfactorily. Histologic verification should be supplied for the working hypothesis that a transient or permanent narrowing of the canal outlets seems to be connected with, or even may become responsible for, the increase in pressure in at least some eyes with primary simple wide angle glaucoma. This factor may be purely hypothetic at this time, but it is possible that it plays an important part in setting the scene for the tragedy of glaucoma. As time goes on, one may expect that the aqueous veins will become a source of further light on the problems of physiology and pathology of the intraocular pressure.

5 West Fourth Street.

News and Notes

EDITED BY DR. W. L. BENEDICT

GENERAL NEWS

Preresident Training Course, Institute of Ophthalmology.—The faculty of Columbia University College of Physicians and Surgeons have approved plans to enlarge the preresident training course (formerly given as the basic science course for the residents of the Institute of Ophthalmology and allied hospitals) so that up to 15 applicants who have received or been promised appointments in institutions approved by Columbia University will be accepted for four months' full time training at the Institute of Ophthalmology.

Anatomy, embryology, pathology, physiological optics, bacteriology, pharmacology, physiology, biochemistry and refraction will constitute the basic studies on which further studies preparing the matriculants for training in ophthalmology will be founded. The course will start Jan. 2, 1950. Applications must be submitted before November 1 to the office of the assistant dean in charge of Graduate Medical Education, 630 West One Hundred and Sixty-Eighth Street, New York 32.

Revised Edition of Motion Picture Reviews Now Available.—The Committee on Medical Motion Pictures has completed the first revised edition of the booklet entitled "Reviews of Medical Motion Pictures." It now contains all the film reviews published in *The Journal of the American Medical Association* up to Jan. 1, 1949. It also includes a classified table of contents, as well as a list of motion pictures available through the Motion Picture Library of the American Medical Association.

The purpose of the reviews is to provide a brief description and evaluation of motion pictures which are available to the medical profession. Each film is reviewed and commented on by competent authorities.

Copies are available on request from the Committee on Medical Motion Pictures, American Medical Association, 535 North Dearborn Street, Chicago 10.

Gifts for Medical Library of University of Chile.—The National Committee for Chile is now receiving gifts for the library of the medical school of the University of Chile at its new collection center in the Library of Congress, Washington, D. C. The newer materials in the library, including periodicals, books and reference materials, were totally destroyed in the recent fire. Medical periodicals of the last ten years and recent medical books are urgently needed. Your contribution will be appreciated.

National Committee for Chile, room 318, Library of Congress, Washington, D. C.

SOCIETY NEWS

Pennsylvania Academy of Ophthalmology and Otolaryngology.—The Pennsylvania Academy of Ophthalmology and Otolaryngology held its annual meeting April 22, 23 and 24 at Harrisburg, Pa.

An interesting program of formal scientific presentations was given, and a round table discussion on "Headache" was presented by specialists in the fields of otolaryngology, ophthalmology, internal medicine and neuropsychiatry.

The following officers of the organization were elected for the ensuing year: president, Dr. Daniel S. DeStio, Pittsburgh; president elect, Dr. Jan G. Linn, Pittsburgh; secretary, Dr. B. F. Souders, M.D., Reading; treasurer, Dr. Bruce A. Grove, M.D., York.

Joint Meeting of Pan-American Association of Ophthalmology and National Society for Prevention of Blindness.—A joint meeting of the Pan-American Association of Ophthalmology and the National Society for the Prevention of Blindness will be held March 26 to 30, 1950. This will be the annual meeting of the latter society and an interim meeting of the former. The headquarters of the meeting will be at the Floridian Hotel, Miami Beach, Fla.

PERSONAL NEWS

Dr. Brittain Ford Payne to Lecture in México, D.F.—In México, D.F., August 15 to 27, Dr. Brittain Ford Payne, clinical professor of ophthalmology at New York University Medical College and director of pathology at New York Eye and Ear Infirmary, will give a course of lectures and demonstrations on the histopathology of the eye. During the first week, after a lecture on general considerations and on the stages in the development of the eye, Dr. Payne will discuss in detail the microscopic anatomy of the cornea, sclera and lamina cribrosa (August 16); the choroid, pigment epithelium, retina and optic nerve (August 17), and the ciliary body and iris (August 18). He will close the first week with a lecture on the normal crystalline lens, the zonular fibers, the chambers of the eyeball and the blood vessels and nerves.

Subjects of lectures of the second week will include diseases of the cornea, sclera and anterior segment (August 22); granulomas of the eye, including tuberculosis and sympathetic ophthalmitis (August 23); glaucoma and cataract, and complications following intraocular operations (August 24); traumatic lesions of the eye, including contusions, perforating wounds and intraocular foreign bodies (August 25), intraocular neoplasms and summary (August 26).

Obituaries

JOHN GREEN, M.D.

1873-1949

Descended from a notable line of physicians and scientists in England and New England, Dr. John Green gallantly accepted the challenge and became justly distinguished by his own diligence and skill in the practice of ophthalmology and by his devotion to public welfare. An early manifestation of the latter service is recorded by his signature as a founder and subsequent activities in behalf of the Missouri Association for the Blind—now the St. Louis Society for the Blind. His many contributions to ophthalmic literature demonstrated a keen analysis and insight into the intricacies of his chosen field of endeavor, as well as a determination to seek the truth and stand by it regardless of consequences.

A résumé of his professional career, as noted in the *Annual Bulletin* of the St. Louis University School of Medicine, where he served as professor of ophthalmology from 1927 until his untimely death, follows: Bachelor of Arts, Harvard University, 1894; Doctor of Medicine, Washington University, School of Medicine, 1898; instructor in ophthalmology, Washington University, School of Medicine, 1912-1919; associate professor of ophthalmology, St. Louis University School of Medicine, 1922-1926. His annual lectures to the senior class and his conferences with ophthalmic residents were carefully prepared and well received. His hospital services were much more extensive and time consuming than this simple résumé can reveal: intern, St. Louis City Hospital (1898-1899); visiting ophthalmologist, St. Louis Maternity Hospital (1910-1926); associate ophthalmologist, Barnes Hospital (1916-1918) and St. Louis Children's Hospital (1916-1918); ophthalmologist, St. Luke's Hospital (1920-1949), St. Mary's Group of Hospitals (1924-1949), De Paul Hospital (1934-1949); ophthalmologist in chief, St. Louis County Hospital (1935-1949); associate ophthalmologist, St. Louis City Hospital (1924-1926); ophthalmologist (1926-1935); consultant (1935-1949). In addition he found time to give unlimited valuable service as member and officer of the American Board for Ophthalmic Examinations and on the advisory staff of the National Society for the Prevention of Blindness. In both world wars he was a busy member of draft boards. He was co-founder and president of the St. Louis Ophthalmic Society; chairman of the Section on Ophthalmology of the American Medical Association; vice president of the

Missouri State Medical Association, and president of the American Ophthalmological Society—the oldest of its kind in the world—as his father had been in the previous century.

He could not be an indifferent member in any of the numerous organizations that claimed his attention, but it would not be hard to prove that his greatest interest was centered in a home at 243 Westgate, near the western city limits of St. Louis. The intangible treasures of home he bequeaths unsullied to his widow, three sons and three daughters and six grandchildren, who brought him life's greatest charm. Only a great man in mind and soul and body could leave such a record. It was his joy to carry the torch for human progress without weakness until his dying day.

W. H. LUEDDE, M.D.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

THE RETICULO-ENDOTHELIAL SYSTEM OF THE CORNEA. T. KRWAWICZ, Brit. J. Ophth. **31**: 421 (July) 1947.

This series of experiments, in which a 1 per cent solution of silver nitrate was applied to the conjunctival sac of rabbits, proves that corneal stroma cells show distinct storing properties for silver, or colloidopexy, and may in some cases be transformed into typical polyblasts. The opinion that stroma cells correspond to wandering cells in a state of rest seems justified. Corneal stroma cells may thus be classified with elements of the reticuloendothelial system.

The article is illustrated.

W. ZENTMAYER.

Cornea and Sclera

USE OF ANTI-ALLERGIC DRUGS IN THE TREATMENT OF PHLYCTENULAR OPHTHALMIA. C. F. BOWES, Brit. J. Ophth. **32**: 163 (March) 1948.

In 15 typical and active cases of phlyctenular ophthalmia, the photophobia, lacrimation, irritation and eczema showed no improvement with diphenhydramine hydrochloride (benadryl®) and 2-(N-phenyl-N-benzylaminomethyl) imidazoline (antistin®), two well recognized antiallergic drugs. All the patients showed positive reactions in the Mantou tests and roentgen ray evidence of tuberculous infection.

W. ZENTMAYER.

A CASE OF KERATOMALACIA CURED BY PENICILLIN AND VITAMIN A. K. RIZK, Brit. J. Ophth. **32**:416 (July) 1948.

The patient, a rachitic, emaciated boy aged 2 years, had had a febrile attack, after which both corneas became opaque. The conjunctivas were dry and showed xerotic spots. There were no signs of inflammation or discharge. The corneas became ectatic and ulcerated. Treatment consisted in the local and general administration of penicillin and a preparation of vitamin A. Vitamin therapy was given in the form of an ointment containing 20 per cent haliver oil and intramuscular injections of a vitamin A preparation ("prepalin"). In five weeks a dense central leukoma covered the pupillary area; the rest of the cornea was clear.

W. ZENTMAYER.

A CASE OF PYOCYANEUS RING ABSCESS OF THE CORNEA TREATED WITH STREPTOMYCIN. J. MASCHLER, Brit. J. Ophth. **32**: 426 (July) 1948.

The cornea of a boy aged 15 was injured by a splinter of iron. When the patient was seen by the author, on the third day, all but a

1 mm. zone of the periphery of the cornea was infiltrated, the central portion being least involved. A greenish blue hypopyon was present; subconjunctival injections of penicillin into the anterior chamber were used provisionally, and later similar injections of streptomycin were given. On the third day of the use of streptomycin, a Saemisch section was made. Two days later regression set in. A leukoma resulted. Secondary glaucoma supervened, for which an iridectomy was done. The bacterial diagnosis was confirmed by culture. The growth of the bacteria was checked with 500 units of streptomycin.

W. ZENTMAYER.

HISTOLOGICAL ASPECTS OF REFRIGERATED CORNEAL EPITHELIUM. R. SEIDENARI, Ann. di ottal. e clin. ocul. **72:** 298, 1946.

On the basis of his histologic researches on refrigerated limbal tissue, the author states that the action of cold on the corneal epithelium results in the following phenomena: (1) slight reduction of the total height of the corneal epithelium, (2) slight and somewhat retarded separation of its superficial layers and (3) good and lasting preservation of the turgescence of the basal layer of cells, with excellent preservation of the histochemica properties of the cytoplasm and of the nucleus. Bowman's membrane showed no changes.

G. B. BIETTI—J. J. LO-PRESTI.

A CORNEAL TRANSPLANT: HISTOLOGIC EXAMINATION. O. VON FIEANDT, Acta ophth. **22:** 36, 1944.

A feature of interest in the case reported was the formation of a cyst in the border of the iris. It was considered an implantation cyst, epithelial cells having been introduced by the trephine at the time of the corneal transplantation.

O. P. PERKINS.

General

THE TEACHING OF OPHTHALMOLOGY. D. VAIL, Am. J. Ophth. **31:** 535 (May) 1948.

Vail, in this Gradle Lecture, discusses qualifications and facilities for teaching, undergraduate and graduate instruction and residency training and aids to teaching.

W. S. REESE.

MILITARY OPHTHALMOLOGY IN THE EUROPEAN THEATER. D. MARSHALL, Am. J. Ophth. **31:** 545 (May) 1948.

Marshall recounts the ophthalmologic work of the 298th General Hospital in Bristol, England, at Cherbourg, Normandy, and Liége, Belgium, particularly from the standpoint of military necessity. He also discusses interesting problems and experiences.

W. S. REESE.

General Diseases

DIABETIC RETINOPATHY. E. REDSLOB, Ann. d'ocul. **181:** 129 (March) 1948.

The author asks two questions: Is it the life-saving insulin that eventually deprives diabetic patients of their vision, or is the retinopathy

a complication of diabetes which arises independently of the glycosuria and hyperglycemia and escapes the therapeutic action of insulin?

Reference is made to published reports on the incidence of diabetic retinopathy. Despite the fact that the incidence appears to be increasing, the author does not believe that the use of insulin should be discontinued. He does not agree that insulin aggravates the condition.

There is no question that the retinopathy is on a vascular basis—renal, arteriosclerotic or hypertensive. The author believes that insulin has a deleterious effect on the vessels themselves through the intermediary of hypoglycemia. Great care must be taken to insure that hypoglycemia does not occur.

Other factors to be considered are the age of the patient and the duration of the diabetes. The occurrence of arteriosclerosis and hypertension with diabetes is discussed in detail, as well as the possibility of renal damage. The author concludes that the intercapillary glomerulosclerosis which occurs in diabetes is probably responsible for the retinopathy. The renal changes may be present for a long time before there is any evidence of renal damage.

These pathologic changes may in time give rise to hypertension and arteriosclerosis. However, the presence of intercapillary glomerulosclerosis may produce diabetic retinopathy in the absence of any clinical manifestations of hypertension, arteriosclerosis or renal damage. The glomerulosclerosis does not depend on the metabolism of the hydrocarbons as manifested by hyperglycemia and glycosuria. It is probable that diabetes and the glomerulosclerosis have a common origin but that the diabetic retinopathy is not a result of the hyperglycemia.

P. R. McDONALD.

CORNEAL LESION ASSOCIATED WITH DUHRING DISEASE. I. ANGIUS, Rassegna ital. d'ottal. **15:** 290, 1946.

The author reports a case of Duhring's disease with the typical manifestation on the extensor surfaces of the extremities and a corneal ulcer in the right eye. During the regression of the ulcer a vesicle formed near it, disappearing in four days without rupture; the appearance of the vesicle was accompanied with the formation of numerous bullae in the skin. The corneal lesions disappeared without residual opacity. There was absence of any conjunctival change. Hyposensitivity of the cornea was noted.

This case illustrates that corneal lesions exist early, that the cornea is not always spared of bullae and that pemphigus and shinking of the conjunctiva are two separate entities. The corneal hyposensitivity may indicate a neurotropic origin, already suggested by Duhring as the basis for the dermatitis. The benign course distinguishes it from ocular pemphigus as a separate disease entity.

G. B. BIETTI—J. J. LO-PRESTI.

THE FUNDUS OCULI IN SEVENTY-EIGHT CASES OF CARDIOVASCULAR DISEASE, IN THE AUTOPSY. C. ESPILDORA LUQUE and A. SCHWEITER, Arch. chilenos de oftal. **15:** 297 (Nov.-Dec.) 1946.

The authors made pathologic studies on 78 subjects who died of various types of renocardiovascular disease and whose eyes had been

examined ophthalmoscopically during life. Twenty-two of the patients were less than 40 years of age. In 7, or 32 per cent, of these young patients the fundus findings were normal. Of the patients over 40 years of age, only 7, or 12 per cent, showed no abnormalities of the fundus. Of the entire series, the fundi were normal in 18 per cent.

The normal appearance of the fundus in both groups of patients was always accompanied with serious lesions of the renocardiovascular system. The renal lesions predominated in the younger group, whereas in the older group the aortic, myocardial, cerebral and renal changes were equally conspicuous.

Usually in the young patients with cardiovascular disease the lesions in the fundus are not pronounced, and a criterion of the general pathologic process and prognosis is not obtained by ophthalmoscopy. An exception occurs in cases of rapidly progressive angionephrosclerosis, in which the changes in the fundus are always severe and pronounced. In spite of the relative normality or slight involvement of the fundus, death in young patients with cardiovascular disease occurs as a result of the serious general involvement.

H. F. CARRASQUILLO.

Glaucoma

CYCLODIALYSIS: RESULTS. V. SADIKOVA, Vestnik oftal. **25:** 42, 1946.

An analysis is made of 93 cases (47 of males and 46 of females) in which cyclodialysis was performed; of these, there were 7 cases of compensated and 87 cases of decompensated glaucoma, 2 cases of absolute glaucoma and 2 cases of juvenile glaucoma. The anterior chamber was shallow in 70 cases, moderately shallow in 21 cases and deep in 9 cases. In 43, or nearly half of the cases hyphema was observed during the operation. In most of the cases the blood was absorbed in three to four days; in 12 cases it was absorbed in two weeks. Prolonged presence of blood in the anterior chamber led to an increase of tension in 7 cases. Postoperative iridocyclitis developed in 7 cases. In 1 case the vitreous prolapsed in the wound.

Evaluation of the results of this operation with respect to vision, intraocular pressure and visual fields was as follows: Vision showed improvement in 41 cases and decrease in 13 cases. The intraocular tension was lowered in all but 7 cases. The decrease in tension was gradual. The visual fields were more contracted (from 5 to 20 degrees) after the operation in 12 cases. In 25 cases there was improvement of the visual fields of from 5 to 25 degrees and in 3 cases of 30 degrees.

Thus, the results were encouraging, and Sadikova states the belief that best results are obtained in simple glaucoma in the early stages.

O. SITCHEVSKA.

HOLTH'S IRIDENCLEISIS. P. RIISE, Acta ophth. **21:** 31, 1944.

The author tabulates his ten years' experience with the iridencleisis operation. A total of 235 eyes, representing glaucoma simplex, secondary glaucoma and acute glaucoma were operated upon. He believes that the operation gives reliable and lasting reduction of tension and few complications.

O. P. PERKINS.

Injuries

SPONTANEOUS EXPULSION OF NUMEROUS WOODEN FRAGMENTS FROM THE EYE: REPORT OF A CASE. E. BOCHEVER, Vestnik oftal. **26:** 36, 1947.

The interest of this case is the spontaneous expulsion of several wooden fragments from the eye within a period of two years. A soldier aged 29 was injured by fragments from an exploded mine. The left eye was enucleated at the front line hospital two days after the injury. Six months later, in the hospital, he presented an irritated right eye, with a corneoscleral scar at 9 o'clock and partial cataract and organized vitreous exudate. The roentgenogram showed no intraocular foreign body. However, at intervals of a few months there would appear a bulging at the scar of the sclera or conjunctiva, and on incision one or two wooden fragments could be extracted at a time.

At the end of two years the result was a quiet eye; the cataract had absorbed, the vitreous had cleared and vision was corrected to a fair amount with a cataract lens. Thus the function of the eyeball was preserved despite the numerous traumas during two years.

O. SITCHEVSKA.

Lens

EXTRACTION OF CATARACT IN THE PRESENCE OF FLUID VITREOUS. D. B. KIRBY, Am. J. Ophth. **31:** 585 (May) 1948.

Kirby discusses fluid vitreous in cases of cataract, with particular attention to its recognition. He believes that these cataracts should be removed in the capsule and feels that preplaced sutures are unnecessary. He reports 10 cases.

W. S. REESE.

FAMILIAL CATARACT WITH EXTENSIVE PEDIGREE CHART. I. L. JOHNSTONE, Brit. J. Ophth. **31:** 385 (July) 1947.

The first family studied consisted of 73 persons, 36 males and 37 females. Of the 73, 23 persons, 13 males and 10 females, were affected. The second family consisted of 58 persons, 28 males and 28 females; the sex of 2 was not ascertained.

Distinctive features of the cataract were (*a*) its presenile onset; (*b*) its position, primarily posterior, subcapsular, and therefore saucer shaped with concavity directed forward, the cataract thus somewhat resembling cataracta aparathyroidea and ray cataract; (*c*) its configuration, with leaflike or feather-like branching radiating from the center, so that it somewhat resembled concussion cataract; (*d*) flakes and dots in white, yellow, green or blue throughout the cortex, but always separated from the anterior capsule by a subcapsular clear zone of disjunction, and (*e*) the accentuation of the shagreen of the anterior capsule. The earliest signs so far observed were visible only with the slit lamp.

An extensive new pedigree of familial cataract with a mendelian dominant method of inheritance is presented, and the biomicroscopic appearance of the cataract is described. The possibility of other causal

factors is discussed with the help of a review of the relevant literature, and a small series of blood analyses is presented.

The article is illustrated.

W. ZENTMAYER.

Neurology

CHRONIC SEROUS MENINGITIS. O. PEDICO, Rassegna ital. d'ottal. **15:** 319, 1946.

The author describes a case of recurrent headache, bilateral papilledema and concentric constriction of the visual field for white and colors. Visual acuity was 20/20 without correction. This spinal fluid showed elevated pressure, was clear and contained 15 cells per cubic millimeter (lymphocytes, 95 per cent; monocytes, 1 per cent, and polymorphonuclear leukocytes, 4 per cent); the albumin measured 0.3 Gm. per hundred cubic centimeters. The author discusses the differential diagnosis and states that the condition in this case was a form of serous ependymitis of Ayala, or chronic serous meningitis.

G. B. BIETTI—J. J. LO-PRESTI.

Nystagmus

CLINICAL REPORT ON MINERS' NYSTAGMUS. D. A. CAMPBELL, Brit. J. Ophth. **32:** 193 (April) 1948.

The report is based on the author's observation on 44 patients with miners' nystagmus. Patients with this condition fall into three grades: patients with actual nystagmus, but no symptoms, who continue to work underground; patients who come within the act for certification, who have headache, photophobia or giddiness and who complain that oscillation of lights underground prevents them from working, and patients with psychoneurosis, who, in addition to the usual symptoms, have blepharospasm whenever they are subjected to examination.

The want of treatment and the lack of occupation after certification may easily convert a man of the second category into the third and produce further deterioration in patients of the third grade.

The miner should return to suitable work, e. g., on the surface, as soon as possible—preferably without an interval of unemployment. Fitness for work should be judged by an ophthalmic surgeon and a psychiatrist with special knowledge of the disease.

W. ZENTMAYER.

THE DARK ADAPTATION OF COAL MINERS SUFFERING FROM NYSTAGMUS. F. W. SHARPLEY, Brit. J. Ophth. **32:** 199 (April) 1948.

Sharpley concludes that the evidence from biochemical tests, although not dramatic, demonstrates that the raised threshold for dark adaptation which is a constant feature among miners, including those with nystagmus, is not due to a lack of vitamin A or D or to any obvious nutritional disturbance.

W. ZENTMAYER.

THE PSYCHIATRIC ASPECT OF MINERS' NYSTAGMUS. E. S. STERN, Brit. J. Ophth. **32**: 209 (April) 1948.

Stern submits the following recommendation for the management of "miners' nystagmus":

The term miners' nystagmus should no longer be used, but all patients complaining of symptoms which would formerly have been considered under that heading should be referred to a psychiatrist as early as possible for appropriate diagnosis and treatment and should be certified as suffering from the actual psychoneurosis or psychosis found.

All such patients should be found work above ground and in the light, instead of being placed on compensation. This work should be found as soon as possible, though a short period of rehabilitation may be necessary, for psychiatric reasons.

The patients should be under psychiatric supervision.

Lighting in the mines should be improved.

Efforts should be made to ease the posture of the miner at work.

Special attention should be paid to safety measures in the mines.

The miner should be given economic security at his work.

Optimum hours of work in the mines should be worked out scientifically, and this matter should be removed from the sphere of politics.

W. ZENTMAYER.

A COMPARISON OF DARK ADAPTATION WITH THE PSYCHOLOGICAL STATE IN MINERS. D. A. CAMPBELL, Brit. J. Ophth. **32**: 225 (April) 1948.

According to Campbell, the evidence, such as it is, indicates that among miners there is no significant relation between the psychologic state and the threshold of dark adaptation. It would be interesting to compare miners with a group of ordinary workmen in this respect.

W. ZENTMAYER.

BINOCULAR VISION IN MINERS. D. A. CAMPBELL, R. HARRISON and J. VERTIGEN, Brit. J. Ophth. **32**: 226 (April) 1948.

The preliminary investigations reported here were made in the hope that a minute study of the behavior of the eyes under conditions of low illumination might reveal the mode of onset of nystagmus.

The binocular vision of miners was compared with that of a large group of normal subjects of the same age groups in full light adaptation and in full dark adaptation (after forty-five minutes in the dark). For subjects able to perform tests for binocular vision, the subjective angle tends to become convergent when they are dark adapted. Miners with nystagmus did not show this phenomenon so strongly. This change did not vary directly with the illumination but appeared to depend on the state of the eye and on the type of visual test. The majority of miners showed an abnormal fixation—their eyes tended to look upward.

The evidence of previous observers is that defective illumination is the primary cause of miners' nystagmus. The authors' observations during the past three years point to a breakdown in binocular vision under conditions of low illumination.

W. ZENTMAYER.

Ocular Muscles

CONGENITAL ABDUCTION DEFICIENCY. P. MATTEUCCI, Rassegna ital. d'ottal. **15:** 345, 1946.

After referring to the confusion that still exists with regard to classification, pathology and pathogenesis of congenital abduction, the author reexamines the problem in the light of observations on 3 patients presenting this syndrome, in the special emphasis on the anomalies and malformations frequently associated with the defect.

In the first case, there were enophthalmos of 5 mm., narrowing of the palpebral fissure and paralysis of abduction (20 degrees). Passive motility was possible in every direction. In the second case there were pernicious anemia, ptosis of the right eyelid, paralysis of abduction of the left eye, with an excursion of 2 to 3 mm., and protrusion of the globe and widening of the palpebral fissure. On looking to the right, the left eye retracted 2 to 3 mm., while the palpebral fissure narrowed and the lid drooped; there was no diplopia. The passive motility test showed some difficulty in extreme abduction of the left eye.

In the third case, there were nephrosis, absence of abduction on either side, retraction on adduction and protrusion on attempts at abduction. The patient died of deficient renal function. Postmortem examination did not reveal any anomalies of insertion of the ocular muscle or presence of anomalous muscles, but atrophy of the external rectus muscles was present. Examination of the cranial cavity revealed nothing of significance except for reduction in size of both abducens nerves, reduction of size and in the number of cells and fibers of the nucleus of the sixth nerve bilaterally and no apparent change in the posterior longitudinal fasciculus. After an exhaustive examination of the literature, the author concludes that the cause of the syndrome lies in a lessened capacity of development (aplasia, hypoplasia and dysplasia).

G. B. BIETTI—J. J. LO-PRESTI.

Operations

AKINESIA. A. KOLEN, Vestnik oftal. **26:** 39, 1947.

Kolen used the following method of producing akinesia for the past ten years. The injection of 1 to 1.5 cc. of procaine hydrochloride 2 per cent with epinephrine hydrochloride 1:25,000 was begun below the lower margin of the orbital wall 1 to 1.5 cm. temporally to the lateral margin of the orbital wall and continued upward on a vertical line 1 to 1.5 cm. above the upper orbital wall. About 2 cc. of the solution is introduced.

Kolen states that this long vertical line of injection insures full anesthesia of all the branches innervating the palpebral orbicularis muscle and that no edema of the upper lid is encountered, such as occurs in van Lint's akinesia.

O. SITCHEVSKA.

Orbit, Eyeball and Accessory Sinuses Parasites

THE FAVORABLE ACTION OF ESTRONE IN CERTAIN CASES OF EXOPHTHALMIC GOITER. J. LEDERER, Am. d'ocul. **181:** 37 (Jan.) 1948.

The author reviews the pathologic physiology of exophthalmic goiter. The production of exophthalmos in animals with preparations of the

anterior lobe of the pituitary is discussed in detail. Clinical and experimental evidence is presented to substantiate the modern concept that the thyrotropic hormone of the pituitary, and not the thyrotoxic hormone of the thyroid, causes the exophthalmos. In some cases the exophthalmos is aggravated by thyroidectomy, and may even be improved by the taking of thyroid. The author was impressed by the number of patients in whom exophthalmic goiter develops at the menopause or after castration.

Five cases of exophthalmic goiter following the menopause or castration are presented in detail. In all cases treatment with estrogenic preparations gave satisfactory amelioration of symptoms and regression of the exophthalmos.

The author believes that the action of the ovarian substances is through the anterior lobe of the pituitary gland. Estrone (folliculin) has an antithyroid action through the intermediary action of the anterior lobe of the pituitary. This checking of the thyrotropic hormone leads to amelioration of the symptoms.

P. R. McDONALD.

Pharmacology

THE ACTION OF BETAPHENYL-ISOPROPYLAMINE ON THE HUMAN EYE. G. SCUDERA, Rassegna ital. d'ottal. **15:** 94, 1946.

The author conducted experiments on human subjects analogous to those already performed on rabbits in order to study the mydriatic effect of amphetamine in various solutions isotonic with blood. The pupillary diameter obtained with solutions of the drug in sodium nitrate averaged 4.3 mm.; with sodium fluoride, 4 mm., with sodium oxalate and with sucrose, 3.5 mm., and, finally, with sodium and magnesium sulfate, 3 mm.

Mydriasis began in fifteen minutes, was maximum after twenty to forty minutes and disappeared in six to eight hours. No notable irritation, change in tension or change in the caliber of the retinal vessels or of the accommodation was observed.

G. B. BIETTI—J. J. LO-PRESTI.

The Pupil

VARIATION OF PUPIL SIZE WITH CHANGE IN THE ANGLE AT WHICH THE LIGHT STIMULUS STRIKES THE RETINA. K. H. SPRING and W. S. STILES, Brit. J. Ophth. **32:** 340 (June) 1948.

Variation in pupillary size depending on the part of the natural pupil through which the light from the external field enters the eye may occur for two reasons: 1. Since the apparent brightness of an illuminated field is reduced when the rays enter near the edge rather than near the center of the pupil (directional sensitivity of the retina), a slightly larger pupil might be expected in the former condition. 2. If, as is possible, the protective action of the constricted pupil consists not so much in reducing the total light flux as in excluding rays which would otherwise strike the retina obliquely, such rays might prove more effective in closing the pupil than would rays of normal incidence. The pupil would then be smaller for rays entering near the edge.

It may be concluded from the present measurements (*a*) that rays entering near the edge of the dilated pupil, and hence incident on the retina at any angle with the normal, do not produce an abnormally high pupillomotor effect and (*b*) that, for practical purposes, the part of the pupil through which light enters the eye is not a factor in determining the size of the pupil.

W. ZENTMAYER.

Refraction and Accommodation

THE FITTING OF CONTACT LENSES. A. MAGITOT, Ann. d'ocul. **180**: 430 (July) 1947.

The author describes the various methods employed in the fitting of contact lenses. Patients vary greatly in their tolerance to the wearing of the lenses. Patients with high astigmatic errors or keratoconus frequently wear their lenses for twelve to eighteen hours. There is a small group, about 10 per cent of the patients, who cannot tolerate contact lenses at all. About 80 per cent of all patients who have contact lenses are able to wear them comfortably for several hours.

The author believes that a variation in the amount of tear secretion may account for some of the differences in tolerance. Mention is made of the phenomenon of Sattler. This is a temporary clouding of the cornea that occurs after the lenses have been worn for four to six hours. It disappears rather rapidly. The author believes that it is caused by an abnormality or a deficiency of lacrimal secretion.

P. R. MCDONALD.

Retina and Optic Nerve

THE ALTERATION IN SIZE OF THE NORMAL OPTIC DISK CUP. R. PICKARD, Brit. J. Ophth. **32**: 355 (June) 1948.

In the present article, 234 eyes observed for fifteen years, or nearly so, are compared with 280 eyes observed contemporaneously with the fifteen year group, but observed only once, and not included in that group. Finally, these two groups are combined, and a general rule as to average growth is deduced. All the disks recorded were normal in appearance. To estimate the size of the cup, a grid was used. The apparent size of the disk and of the cup surface are found, and the size of the cup is expressed as a percentage of the disk. Any cup of a size above 70 per cent should be recorded as suspect and be considered with respect to the visual fields and tension before it can be said to be normal. In 1 eye the cup was 10 per cent smaller at the end of fifteen years. Twelve others showed a decrease at some time, though all these were larger at the end of the fifteen years than at the beginning. One cup was more stationary at the end of the fifteen years than at the beginning. One cup was stationary; it was 36 per cent of the disk. It is not uncommon to have a period of arrest of growth of the cup. Increase in the depth tends to occur with the enlargement of the cup area, but irregularly so. It may be stated as a generalization that if a cup enlarges to 60 per cent of the disk, its depth will increase generally by 0.5 to 1.0 D. A disk may increase to 100 per cent and yet retain a depth of 1.0 D. An increase to 3.0 D. with an increase in the cup percentage

must be investigated by determination of the field and the tension. The possible causes of enlargement of the cup are discussed.

W. ZENTMAYER.

RETINAL ARTERIAL PRESSURE AFTER LUMBAR PUNCTURE. M. MECCA, Ann. di ottal. e clin. ocul. **71:** 336, 1946.

The author reviews the various theories as to the mechanism by which changes in pressure in the retinal arteries are produced by variations in the intracranial pressure. He reports the results of several studies on the behavior of spinal fluid pressure and intracranial pressure after lumbar puncture. He shows that after lumbar puncture the following changes occur: (1) an immediate rise in the retinal arterial pressure, and (2) a slight drop to the initial level after twenty-four hours. The initial rise is an expression of the shift of intracranial pressure produced by the lumbar puncture, and both these changes, according to the author, are related to a vascular phenomenon.

G. B. BIETTI—J. J. LO-PRESTI.

TWO CASES OF ABLATIO RETINAE CONGENITA NONFALCIFORMIS. H. MOLLER, Acta ophth. **23:** 353, 1945.

Bilateral detachments of the retina were observed in a boy of 5 years. No tears or other changes were found. One eye was removed because the presence of a malignant growth was suspected. None was found. Later examination of a younger brother, aged 2, showed a similar condition in one eye.

O. P. PERKINS.

Tumors

CONJUNCTIVAL NEVI. D. ARCURI, Rassegna ital. d'ottal. **15:** 263, 1946.

The author discusses conjunctival nevi from a clinical standpoint by referring to the literature and by reporting 2 of his own cases. He stresses the importance of slit lamp examination, particularly with respect to the vascularization present. This examination may well be substituted for biopsy, which is technically more difficult and is not free from danger. The stationary nevus should be left undisturbed, while any nevus which increases in size should be freely excised surgically and followed by diathermy coagulation.

G. B. BIETTI—J. J. LO-PRESTI.

CONGENITAL SCLEROCORNEAL CYST: REPORT OF A CASE. V. BISCHLET, Ophthalmologica **114:** 371 (Oct.-Nov.) 1947.

A case of congenital sclerocorneal cyst is reported. The article is well illustrated. The literature since 1853 is reviewed.

H. P. KIRBER.

Uvea

A CASE OF RECURRENT APHTHOUS UVEITIS WITH ASSOCIATED ULCUS VULVAE ACUTUM (LIPSCHUTZ). A. RUGG-GUNN, Brit. J. Ophth. **31:** 396 (July) 1947.

The patient, a married woman aged 22, had had fourteen attacks of the condition, eight in the left eye and six in the right eye. The relapses

were of sudden onset and of relatively short duration. Rigor occurred on several occasions. About eight months after the final attack of uveitis vulval ulceration was noted. There were no dermatologic symptoms. Pregnancy occurred and went on to term. The condition was resistant to penicillin and the sulfonamide compounds.

The evidence seems to point to a virus infection as the most probable explanation of the disease.

The article is illustrated.

W. ZENTMAYER.

CHOROIDAL APOPLEXY DIAGNOSED AS SARCOMA OF THE CHOROID. AUW-YANG SIEN, Ophthalmologica **115:** 1 (Jan.) 1948.

The patient, a woman aged 56, had light perception only in the affected eye. The pupil appeared black on transillumination, and no fundus reflex was obtained. The diagnosis was intraocular pigmented tumor, and the eye was enucleated. Histologic examination revealed apoplexy of the choroid, caused by the rupture of a ciliary artery. The author briefly reviews the literature and points out that there is no way of differentiating clinically between hemorrhage and sarcoma of the choroid.

H. P. KIRBER.

Vision

MONOCULAR OPERATION FOR HIGH DIVERGENT STRABISMUS. L. WEEKERS, Am. J. Ophth. **31:** 541 (May) 1948.

Weekers describes an interesting operation for high degrees of divergent squint in which he attaches the internal rectus to the external rectus muscle of the same eye, thus permitting new scleral attachments for the internal rectus. A number of preoperative and postoperative photographs are shown.

W. S. REESE.

SOME ASPECTS OF STUDYING BINOCULAR VISION. K. C. SWAN, Am. J. Ophth. **31:** 845 (July) 1948.

Swan insists on a summary of a patient's deficiencies and analysis of his binocular potentialities as a basis for therapy and evaluation of treatment. He does not consider the patient normal until he demonstrates comfortable single binocular vision under his usual working conditions.

W. S. REESE.

Visual Tracts and Fields

RECORDING OF VISUAL FIELDS. M. CHAMLIN, Am. J. Ophth. **31:** 565 (May) 1948.

Chamlin describes a method of recording visual fields which permits the examiner to work rapidly, uninterruptedly and accurately.

W. S. REESE.

VALUES AND LIMITS IN COLOR PERIMETRY. G. CRISTINI, Rassegna ital. d'ottal. **15:** 381, 1946.

The author studied a series of patients with diseases of the perceptual and conducting structures in order to collect information on

peripheral color fields. He used (1) chromatic light stimuli (not pigments) of sufficient intensity to obtain a visual field as large as was obtained with white light; (2) light stimuli of equal brilliance and saturation so as to compare the curves obtained, and (3) pure color (monochromatic light). To obtain these stimuli, he used three monochromatic filters from the Leitz photometer placed in front of opal glass and a system of lenses which made parallel rays from a lamp behind the instrument. Photometric determinations were made so as to calibrate the brilliance against changes in the current.

After having reported the limits corresponding to the average values found for 20 normal subjects, the author describes the variations encountered in various ocular disorders: (1) diseases which principally involve the outer layers of the retina (choroiditis, retinitis pigmentosa and detachment of the retina) and inner layers (embolism, thrombosis and glaucoma), and (2) diseases of the conduction apparatus, namely, optic nerve in the region of the chiasm (inflammatory and pressure lesions) and of the geniculocalcarine tract.

A study of the form fields of these patients made at the same time gave following findings: Only in early diseases of the outer layers of the retina was there a disproportion between form fields and color fields, in that amblyopia for blue and green was noted; in diseases of the conducting system, he found no difference between form and color fields so long as the brilliance of the white target and that of the colored target were equal.

The author concludes that the values and the limits of color perimetry in diseases of the conduction system are no different from those obtained by quantitative perimetry.

G. B. Bietti—J. J. Lo-Presti.

Angioscotometry. H. Goldman, Ophthalmologica **114:** 147 (Sept.) 1947.

The author finds that the breadth of an angioscotoma is dependent on the contrast $\Delta I/I$. The angioscotoma becomes wider with diminishing contrast, and always in the direction of the movement of the object. While Evans found that the physiologic angioscotoma increased in a symmetric fashion, with pressure on the eye, pressure on the opposite eye and compression of the jugular vein, Goldman finds that these changes occur, but always in an asymmetric fashion. The angioscotoma increases in breadth in the direction of the movement of the object. This suggests to Goldman that the angioscotoma is not caused by filtration of fluid out of the blood vessels into the surrounding retina, producing a block in conduction, as Evans claimed, but is due to some physiologic property of the retina itself. Further experiments show that other types of scotomas, independent of the retinal vessel tree, show the same characteristics. It is quite apparent, therefore, that the finer so-called angioscotoma is actually a phenomenon of summation, as a result of the rods and cones being connected outside the foveal area with more than one cell station higher up in the synaptic pathway

to the brain. This relationship permits summation and accounts for the appearance and disappearance of the scotoma when the stimulus is subliminal.

F. H. ADLER.

Vitreous

OX VITREOUS HUMOR: I. THE RESIDUAL PROTEIN. A. PIRIE, G. SCHMIDT and J. W. WATERS, Brit. J. Ophth. **32:** 321 (June) 1948.

On the basis of the experimental evidence submitted, the authors conclude that the structure of the vitreous humor of the ox is complex. The vitreous cannot be considered uniform but is made up of at least two "structures," the collagen-like network of the hyaluronic acid and protein jelly. Liquefaction of the vitreous follows enzyme action and is essential for maintenance of a normal vitreous; yet it does not form the vitreous, being minimally swollen at the neutral point and occupying only a very small part of the total volume. It seems that the relation of the network to the jelly is of prime importance and that it must be taken into account in any theory of the causes of swelling and degeneration of the vitreous.

The authors summarize their work as follows: Roentgenology, amino acid chromatography and enzyme analysis provide evidence that the residual protein of the vitreous humor of the ox is largely of collagen type. Enzyme preparations containing collagenase liquefy the ox vitreous. The bearing of this observation on the conception of the structure of the vitreous is discussed.

W. ZENTMAYER.

EXPERIENCES WITH TRANSPLANTATION OF HUMAN VITREOUS. O. VON FIEANDT, Ophthalmologica **115:** 257 (May) 1948.

Vitreous taken from human eyes from eight hours to three days after death was transplanted into 4 human eyes. Two of these eyes were ultimately lost, owing to a violent inflammatory reaction. The remaining 2 eyes tolerated the foreign material well, 1 of them on two occasions. In 1 eye a detached retina was found to be well in place after the procedure. The blood groups of the donor and recipient seem to have no influence on the outcome of the operation. The intradermal injection of the donor's vitreous (Hobart test) is not a reliable indicator of hypersensitivity.

H. P. KIRBER.

Therapeutics

ACTION OF PRISCOL® ON THE VESSELS OF THE RETINA. B. MILLER, Ophthalmologica **115:**11 (Jan.) 1948.

Priscol® (2-benzyl-4,5-imidazoline hydrochloride) has been used with good results in treatment of caustic burns of the eye, keratitis and iridocyclitis. The drug acts as a vasodilator on the retinal vessels. Patients with retinal arteriolar spasms were selected for the study. Six to 10 intravenous injections of priscol® 0.01 Gm., were given over a period of fourteen days. Subjective improvement consisted of decrease in fatigue and headaches; objective improvement was evident in widening of the retinal arteries, decrease of blood pressure, and better visual

acuity. The drug acts as a dilator on spastic, as well as on normal, arteries. Measurements were made with the Lobeck method on 10 normal subjects and on 10 subjects with arterial spasm. The results are tabulated. Two cases are described in detail. The first patient, a man aged 66, had thrombosis of a branch of the upper temporal vein in one eye. He also presented moderate congestion of the lungs, acrocyanosis and insufficiency of the coronary vessels. After three weeks of treatment with priscol,® his vision improved from 2/35 to 5/20, and the fields also showed decided improvement. The second patient, a man aged 66 with diabetes and chronic nephritis, showed only moderate improvement of visual acuity and fields but experienced marked subjective improvement in general.

H. P. KIRBER.

Society Transactions

EDITED BY DR. W. L. BENEDICT

OXFORD OPHTHALMOLOGICAL CONGRESS

H. B. Stallard, M.B.

Thirty-Third Annual Congress, July 3, 4 and 5, 1947, London

The Thirty-Third Annual Oxford Ophthalmological Congress was held July 3, 4 and 5, 1947.

The Doyne Memorial Lecture was admirably delivered by Dr. Leon Stansfield Stone, Bronson Professor of Comparative Anatomy, Yale University, on the subject "Return of Vision and Functional Polarization in Retinas of Transplanted Eyes."

Salamander eyes offer unique opportunities for developmental and regenerative studies of the retina and optic nerve fibers. Long before the sensory layers in the retina are even differentiated it is possible to uncover the moment at which the future functional patterns in the retina are determined. This is borne out by the observation that when the embryonic eye is rotated 180 degrees subsequent to the late tail bud stages the visuomotor responses are completely reversed in the larval and adult life of these hosts. A similar operation earlier than this period in development is followed only by normal vision.

Although the retina survives in transplanted larval eyes, it degenerates and then regenerates in adult grafts. After transplantation in the adult eye the functional patterns in the retinal quadrants can be made to reestablish themselves in the new retina. Vision can return four times in the same adult salamander eye repeatedly transplanted to a new host. The eye can also be successfully transplanted after seven days of refrigeration.

Normal visuomotor responses are easily demonstrated as the animal follows a lure approaching in all directions. The animal also automatically follows vertical black and white stripes in a drum rotating in one direction only (temporonasally) through the field of vision of an eye (clockwise for a left eye; counterclockwise for a right eye).

If the grafted eye is normally oriented, visuomotor responses are normal. If the excised eye is reimplanted upside down (all retinal quadrants rotated 180 degrees), abnormal swimming reactions and reversed visuomotor responses are established after the new retina and optic nerve regenerate. The effect is the same as in the control experiment, in which the normal eye is rotated 180 degrees in situ, with preservation of the original retina and nerve. In either case normal vision is restored immediately by rotating the eyes in situ back to normal position without injury to the retina or nerve.

The dorsoventral and nasotemporal axes may be independently rotated 180 degrees in a single eye by exchanging the right and the left eye and properly orienting them. Only through the reversed retinal quadrants are the visuomotor responses reversed. Rotated eyes grafted to animals of different species give similar results. The abnormal visuo-

motor responses have remained established in animals kept alive for three and one-half years. The application of these studies to visual acuity and color vision was discussed, and a colored motion picture, illustrating with beautiful precision the results of these investigations, was shown.

This excellent lecture was immensely appreciated by the large audience present.

The discussion on "The Contracted Socket" was opened by Prof. T. Pomfret Kilner and Mr. H. B. Stallard. Professor Kilner discussed the causes of contraction and commented on the suddenness with which this occurred in some cases. He expressed himself in favor of using a very thin skin graft (Thiersch). He showed a variety of operative prostheses intended to produce overcorrection and to allow for subsequent contraction of the socket. Retention of these "forms" was by central tarsorrhaphy. Professor Kilner discussed the treatment of unpleasant odor from skin graft sockets by swabbing with "cetavlon" (cetyl trimethyl ammonium bromide) and grease massage. He showed lantern slides of treated cases and a cinema of the standard technic used in his clinic at Roehampton.

Mr. Stallard described the anatomic requirements of the ideal socket and the common defects and deformities seen in contracted sockets. He discussed the mechanical enlargement of some contracted sockets by graduated sizes of acrylic molds, held, if necessary, by a retention device consisting of a screw bolt buried in an acrylic mold and attached by three pins and two universal joints to a plate buried in a plaster of paris head band.

He described his technic of conserving palpebral conjunctiva and using full thickness skin grafts cut to a special pattern to fit the concave under surface of an acrylic mold, which was shaped to the ideal size of the prosthesis ultimately required for the patient. The acrylic mold was perforated with two central holes 3 mm. in diameter for the escape of discharges from the socket. The free skin graft was sutured to the edges of the conjunctiva in the upper and lower fornices and at the medial and lateral canthi.

To date, such grafts had taken completely; a good projecting mound was formed in the socket floor to fill the concavity of the ultimate prosthesis; there had been no foul discharge or smell and no obvious contraction of the graft. It was stated that such work was still in the experimental stage and that it was too early to make a definite recommendation for the acceptance of this technic. The results showed, however, that the marriage of skin and conjunctiva was not always an unhappy one.

Mr. Stallard commented that the full thickness graft was tried because the Thiersch grafts always underwent considerable contraction.

Miss Savory (London), in her paper entitled "Uses of Thrombin and Fibrinogen in Ophthalmic Surgery," spoke of the importance of using higher concentrations of fibrinogen and thrombin than are normally present in the blood in order to produce firmer and quicker clotting. Human fibrinogen is used in a 1.5 per cent solution (the normal fibrinogen content of the blood is 0.25 per cent), and thrombin in a concentration of 5 units per milliliter. First, the graft bed and the deep surface of pedicle grafts are flooded with fibrinogen, and later 2 or 3

drops of thrombin are added. The graft is spread into its proper position at once.

In the case of free mucous membrane and epidermal grafts applied to the eye and socket, sutures are necessary, and these are placed before fibrinogen and thrombin are applied, through a lacrimal cannula inserted beneath the graft.

These substances are not satisfactory for conjunctival flaps reflected in the surgical repair of squint and retinal detachment, but are effective in sealing conjunctival flaps in the classic cataract extraction operation and the torn edges of lacerated wounds of the conjunctiva.

The fibrin clot, tinted with methylene blue, is also used in distending the lacrimal sac for excisions. Thrombin, 5 units per cubic centimeter, has been injected into the anterior chamber to prevent hyphema in cases of new vessel formation on the iris and to wash out the anterior chamber after evacuation of the blood in cases of recurrent hyphema.

Mr. Tudor Thomas described his technic of obtaining by a trephine donor corneal grafts from cadavers, leaving the rest of the eye in situ.

Mr. George Black gave an account of the diagnostic value of injecting methylene blue, 3 minims (0.18 cc.) of a 0.1 per cent solution, beneath the retina. The sclera is trephined at the site for injection. The detached area of the retina rapidly stains and tears show up vividly as red patches against the blue background. The choroid does not stain.

At the end of the Congress, an excellent and most interesting cinema and demonstration was given of Dr. Norman L. Cutler's operation, a ball and ring implant into Tenon's capsule after excision of the eye.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Brittain F. Payne, M.D., *Chairman*

Milton L. Berliner, M.D., *Secretary*

May 19, 1947

INSTRUCTION HOUR

Common Errors in Fitting Glasses. Mr. Aurel Mangold (by invitation).

Aids to Subnormal Vision. Mr. Conrad Carlson (by invitation).

Plastic Eye Exhibit. Major Stephen F. Smolczynski, D. C., United States Army (by invitation).

PAPERS OF THE EVENING

Heredoretinal Degenerations Associated with Brucellosis. Dr. Edwin M. Burton (by invitation).

Cases of macular degeneration of the adult type occurring in 2 brothers and 1 sister were reported. Each patient gave strongly positive reactions to tests for brucellosis. There was no history of consanguinity among the parents or ancestors or of ocular disease among parents or relatives.

The older brother, aged 35, had some difficulty in reading while in high school, and in 1943 he consulted a physician because of gradual failure of vision. Vision with glasses was 20/30 in the right eye and 20/200 in the left eye, eccentric. Ophthalmoscopic examination revealed normal disks. Numerous yellowish white spots, situated fairly deep in the retina, were scattered throughout the posterior segments. In the right eye the macular region appeared dull and the foveal reflex was absent. In the left eye the macular region was also dull in appearance and a "hole in the macula" was apparently developing. In the right eye there was a relative central scotoma for blue and red, containing a paracentral absolute nucleus. In the left eye there was an absolute central scotoma. Two children, aged 7 and 4 years, were apparently normal in every respect.

The sister, aged 29, noticed that her vision was failing in the fall of 1945. Vision with glasses was 20/30 in the right eye and 20/200 in the left eye. On ophthalmoscopic examination the disks appeared normal. The macula of the right eye appeared swollen, and there was a slight disturbance in pigment and a few small glistening spots in this area. In the left eye the picture was essentially the same, except that the foveal reflex appeared widened. A central scotoma was present in each eye, relative to 1 degree white and absolute to 1 degree blue and red.

The younger brother, aged 31, had recently noted slight disturbance of vision. Objects appeared to quiver in the late afternoon. Vision was 20/20 in the right eye and 20/20 in the left eye. On ophthalmoscopic examination the disks appeared normal, the fovea of each eye dark and the foveal reflex broadened, so that one had the impression that the reflex was situated slightly anterior to the surface of the retina. Surrounding each fovea the retina showed a very narrow, indistinct band of extremely fine stippling. In the right eye there was a small paracentral scotoma, and in the left eye there was a narrow ring scotoma. One child, aged 18 months, was apparently healthy.

The cases represented different stages of the same type of macular degeneration. "Hole in the macula," yellowish spots and stippling of the retina have all been described by other authors. The younger brother probably represents the earliest recognizable stage of the disease. In this case the presence of a broadened, anterior foveal reflex and the narrow ring scotoma may be important points in the early diagnosis of the condition. The influence of infection on the development of similar macular lesions has been suggested by several observers. The role of brucellosis in the development of these lesions is extremely doubtful but is, nevertheless, an interesting coincidence.

DISCUSSION

DR. RALPH I. LLOYD: We are indebted to Dr. Burton for this report of a family with hereditary macular degeneration. Few of these lesions ever reach the laboratory, for only eyes presenting a menace to life or to the other eye, or those too painful to bear, ever reach the pathologist. Knowledge of this disease is limited, and only observations on families over a period of years will compensate for the lack of pathologic material. Photographs or paintings should be made from time to time to show the various stages and aspects, of which present information is scant. I hope Dr. Burton will keep this family under

observation for years and will finally give us a report, including the progeny in his survey. If one can awaken the interest of oculists generally, the disease will be better understood and an occasional eye will be obtained, so that eventually the descriptions in textbooks will measure up to the requirements of instruction in our specialty.

DR. ARNOLD KNAPP: May I add that I had the opportunity of seeing recently, after twenty years, one of my patients with macular degeneration to which Dr. Burton referred. This woman has kept the same vision, and the fundus picture has not in any way changed; she has had a number of children, and their eyes are normal.

Reconstruction of the Upper Lid (a color film). DR. ALSTON CALLAHAN (by invitation), University, Ala.

The preoperative appearance of a man aged 20 with a deformity of the lid since birth was shown. A large central area of the right upper lid was missing, in the manner of a congenital coloboma.

The surgical procedure, performed with the use of local anesthesia, was shown in detail. Both the medial and the lateral fragments were divided into an anterior lamina of skin and orbicularis muscle, and a posterior lamina of tarsus and conjunctiva. Small rectangular areas were denuded on the margins of the upper lid fragments and on the margin of the lower lid to provide for intermarginal adhesions for the maintenance of tension on each side of the defect. The two parts of the posterior lamina were then joined with mattress sutures (chromic surgical gut 0000).

The anterior fragments could not be joined without further relaxation, for the tension would tear out sutures through them. The medial fragment was too short to furnish needed tissue. The lateral fragment was entirely divided into the laminas, and the anterior lamina of the lid was separated from the lateral palpebral raphe by making an oblique incision upward through the skin at the extremity of the lid. The two parts of the anterior lamina were then joined easily with interrupted sutures (braided silk 0000). The intermarginal adhesions were then made by uniting the small rectangular denuded areas with Wheeler sutures.

To correct the surgical defect caused by the medial displacement of the lateral fragment, the area of skin and muscle lateral to the oblique incision was undermined and advanced medial to the transposed lamina. This incision was then closed with interrupted sutures. Penicillin solution (500 units per cubic centimeter) was applied liberally, and the lids of both sides were bandaged firmly.

After five days all dressings were discontinued, and after seven days the sutures were removed. The lid adhesions were allowed to remain for three months to prevent retraction of the fragments or the formation of a notch. After this time they were divided, and the result as seen two months later was satisfactory functionally and cosmetically.

DISCUSSION

DR. WENDELL L. HUGHES: This is a beautiful demonstration of a surgical procedure. I think there is no question about its being a congenital coloboma. In the moving picture, the difference of several

millimeters in the level of the medial canthus on the right and that on the left was very nicely demonstrated. I had the opportunity of operating this afternoon on a baby with a somewhat similar condition in the lower lid, in which the difference in the medial canthus on the affected side was about 5 mm.—an extreme difference. Another child, 4 years old, on whom I operated three weeks ago had a coloboma of the upper lid similar to that illustrated in the moving pictures. There was not so much of a difference in the level of the medial canthus as in the present case. The importance surgically and cosmetically of the two layers in the lid is graphically demonstrated by Dr. Callahan's beautiful presentation.

DR. BYRON SMITH: I should like to ask a question concerning the apparent ptosis. Was that real, or merely due to the light; if it was a real ptosis, what does Dr. Callahan anticipate in the treatment of it?

DR. ALSTON CALLAHAN: The levator palpebrae superioris functions normally, and the apparent ptosis is due to the manner in which the lid was reconstructed. The last photographs shown were made five months after operation. This patient resides in the East and was seen by me only a few days ago, eighteen months after operation. The right lid now more nearly matches the left because of the changes due to the passage of time.

Ocular Leprosy in Panama (motion picture). DR. ROBISON D. HARLEY (by invitation), Atlantic City, N. J.

Ocular leprosy is not uncommon in Panama or in the neighboring countries. All patients known to have the disease are confined in the leprosarium of the Canal Zone, where facilities are at hand for careful study.

In a recent study of 150 cases over a period of four years, ocular complications were found in 90 per cent. Fifty-four per cent of the patients had vision of 6/60 or less, and of this number 13 per cent were blind.

Leprosy affects the anterior segment of the eye almost exclusively. The cornea is the most vulnerable of the ocular tissues and was involved in 58 per cent of the cases. The commonest form of keratitis was a typical "chalk dust," superficial, punctate keratitis, which occurred in 34 per cent of all cases. Interstitial keratitis and deep keratitis were common.

Iritis, or evidence of iritis, was found in 50 per cent of the cases. Pinpoint lepromas engrafted on a chronically inflamed iris were frequently seen with the slit lamp.

The limbus was the favorite site of larger lepromas. Lagophthalmos, due to peripheral involvement of the seventh nerve and ectropion were frequently seen.

Absolute corneal anesthesia was rare, but relative loss of corneal sensitivity does occur in severe cases. The conjunctiva appears to be rather resistant to the disease.

Promin® (sodium p, p'-diaminophenylsulfone-N, N'-didextrose sulfonate) was an effective drug in the treatment of leprosy. Secondary infection cleared, and recurrent attacks of iritis were less common.

DISCUSSION

DR. GEMINIANO DE OCAMPO: I have very little experience with leprosy of the eye. In the Philippines it is true that there are plenty of lepers, but they are segregated in a colony, and we in the department of ophthalmology of the Philippine General Hospital in Manila rarely see a case of leprosy. About ten years ago I examined clinically several patients with lesions of ocular leprosy, but I have not seen them a second time. The disease was of the granulomatous type, affecting the lids and the anterior segment of the eye—the iris, ciliary body, cornea, sclera and episclera.

DR. ARTHUR LINKSZ: What did you do for personal protection?

DR. ROBISON D. HARLEY, Atlantic City, N. J.: First, I was advised to wear gloves and a mask, but the patients resented being treated as untouchables. In order to get the cooperation of these patients for observation and treatment, one must first make friends of them. So I discarded the gloves and mask, and when they saw I was not visibly afraid of them they began to warm up to me. At Palo Seco Leper Colony medical treatment is not compulsory. They attend the dispensary voluntarily. Soon I found the clinic attendance increasing satisfactorily, and I never returned to the gloves and mask.

According to the literature, there have been no cases of doctors or nurses contracting the disease; so I felt relatively safe. The case of Father Damien, on Molokai Island, is one of the few known instances on record in which a normal adult acquired the disease through contact. At the conclusion of the clinic we scrubbed carefully with soap and water and followed with an alcohol wash.

Malignant Melanoma of the Choroid with Sympathetic Ophthalmia. DR. HUNTER ROMAINE (by invitation).

A Russian woman aged 50 had a history of a red, painful right eye of one year's standing. Examination revealed a glaucomatous (tension, 90 mm.), severely inflamed eye with corneal edema, a large swollen lens, poor light projection and restricted transillumination. The lens was extracted, followed by return of tension and posterior uveitis, rather typical of sympathetic ophthalmia, in the left eye, with reduction of vision to 20/400.

Permission was gained for enucleation (three months later). General systemic treatment was followed by clearing of the uveitis, revealing optic neuritis, which, in turn, subsided. Normal vision was restored to the remaining eye.

The author discussed the possibility of symptoms of sympathetic ophthalmia developing from the melanoma or after operation and the probable maintenance of vision if the ophthalmia is the true sympathetic type.

DISCUSSION

DR. BRITTAIN F. PAYNE: A section of this eye was presented at a meeting of the Pathology Club in Washington, D. C., last month. It was studied by every one there, including Dr. Verhoeff, who concurred with Dr. Romaine in the diagnosis of sympathetic ophthalmia in the presence of a tumor. Dr. John Reid had just such a case about

fifteen years ago, with a history of the same type. In his closing remarks, Dr. Romaine has consented to show Dr. Reid's slide and to say a few words about the case.

Dr. Arnold Knapp: How do you differentiate sympathetic ophthalmia arising from traumatic iridocyclitis after a cataract operation and one secondary to a sarcoma of the choroid? I wish to point out that the sympathetic ophthalmia of the second eye, which shows itself in the form of an optic neuritis, is always a benign form.

Dr. Ludwig von Sallmann: Dr. Romaine allowed me to see one of the sections in his case. The changes in the uvea were, in my opinion, consistent with the diagnosis of sympathetic ophthalmia. Since I did not know the surgical history of the case and could not deduce it from the section, I concluded that the sympathetic ophthalmia was connected with the sarcoma.

Dr. Emanuel Rosen: Dr. Rados, who has studied this problem extensively, has pointed out that in the case of sympathetic ophthalmia following a sarcoma of the choroid, provided serial sections are made, one will usually find a perforation of the globe somewhere; so the element of trauma is introduced, as was done surgically here.

Dr. Hunter H. Romaine: Dr. Knapp, I wish I could differentiate the two types of sympathetic ophthalmia. I realize that the etiologic factor, and even the presence of the sympathetic ophthalmia, is controversial in this case; but I thought the case was interesting enough to justify presentation. I wish I were able to make that differentiation.

Dr. John Reid, of Lenox Hill Hospital, has a case in which the eye shows almost the same condition. The patient sustained an injury to the eye and glaucoma developed. A filtration operation was performed, and two weeks later the good eye became inflamed. A diagnosis of sympathetic ophthalmia was made, and on the basis of poor transillumination a tumor was suspected. The exciting eye was removed, and the specimen was shown. The tumor appears somewhat smaller, and it is a little more difficult to make out the uvea. The tumor tissue itself has similarly lined and unlined vessels. The tumor cells are rather difficult to make out, but the same type of infiltration is noted here as in my case; the difference is that endothelioid cells and giant cells could be located in the tissue in his case and not in my case. There is definite lymphocytic infiltration.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Dr. A. Churchill, 66 Boulevard Saint-Michel, Paris, 6e, France.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Council of Ophthalmology

President: Prof. F. W. Nordenson, 3 Grefgatan, Stockholm, Sweden.
Secretary: Dr. Halger Ehlers, Rigshospital, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 17 Horseferry Rd., London S. W., England.
Secretary: Dr. F. Wibaut, P. C. Hoofstraat 145, Amsterdam, Holland.

Pan-American Association of Ophthalmology

President: Dr. Conrad Berens, 301 E. 14th Street, New York 3.
Secretary: Dr. Thomas D. Allen, 122 So. Michigan Ave., Chicago 2, Ill.
Place: Interim Meeting: Miami Beach, Fla. Time: March 26-31, 1950; IV Pan-American Congress of Ophthalmology early in 1952.

Pan-American Association of Ophthalmology
Puerto Rico Chapter

President: Dr. Luis J. Fernandez.
Secretary: Dr. P. Fernandez.
Place: San Juan, Puerto Rico.

FOREIGN

All-India Ophthalmological Society

President: Dr. S. A. Rahim, Kachiguda, Hyderabad (Deccan).
Secretaries: Dr. G. Zachariah, 16 Marshall's Rd., Egmore, Madras. Dr. S. N. Cooper, Laud Mansion, 15 Queen's Rd., Bombay.

Belgian Ophthalmologic Society

President: Dr. L. Weekers, Rue Forgeur 15, Liége.
Secretary-General: Dr. M. Appelmans, Avenue Ruelens 179, Louvain.
Place: Bruxelles. Time: January, June and November.

Bombay Ophthalmologists' Association

President: Rotated
Conveners: Dr. S. N. Cooper and Dr. B. D. Telang, Laud Mansion 21, Queen's Road, Bombay 4.

British Medical Association, Section on Ophthalmology

President: Mr. O. G. Morgan, 115a Harley St. W. 1, London, England.
Secretary: Mr. A. G. Cross, 27 Harley St., London W. 1.

Chengtu Ophthalmological Society

President: Dr. Eugene Chan.
Secretary: Dr. D. S. Shen, Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China.
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd. Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

FOREIGN

Deutsche Ophthalmologische Gesellschaft Heidelberg

President: Professor Dr. med. K. Wessely, München, Universitäts-Augenklinik.
Secretary: Professor Dr. med. E. Engelking, Heidelberg, Universitäts-Augenklinik.
Place: Heidelberg. Time: 18.V.1949.

Faculty of Ophthalmologists

President: Dr. Frank W. Law, 45 Lincoln's Inn Fields, London, W.C. 2, England.
Secretary: Dr. J. H. Doggart, F.R.C.S.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Medical Trade Union, Section of Ophthalmology

President: I. Csapody, János-Kórház, Budapest, Hungary.
Secretary: E. Galla, Krisztina Körut 139, Budapest, Hungary.
Place: Illes u. 15, Budapest, Hungary.

Hungarian Ophthalmological Society

President: Prof. G. Horay, Budapest.
Assistant Secretary: Dr. Stephen de Grósz, University Eye Hospital, No. 1, Illesucca 15, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Israel Ophthalmological Society

President: Dr. Aryeh Feigenbaum, 15 Abyssinian Street, Jerusalem.
Secretary: Dr. E. Sinai, 9 Bialik St., Tel Aviv.

Midland Ophthalmological Society

President: Dr. H. Campbell Orr, 8 Summerfield Road, Wolverhampton, England.
Secretary: Dr. P. Jameson Evans, 51 Calthorpe Rd., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

Nederlandsch Oogheelkundig Gezelschap

President: Prof. A. W. Mulock Houwer, Bussum.
Secretary: Dr. T. A. Vos, Laan v. Meerdervoort 394, the Hague.

North of England Ophthalmological Society

President: Mr. J. S. Arkle, 3, Eslington Road, Newcastle-on-Tyne.
Secretary: Mr. W. M. Muirhead, 70 Upper Hanover Street, Sheffield, 3.
Place: Manchester Leeds Newcastle, Liverpool, Sheffield & Bradford.
Time: October to May.

Ophthalmological and Oto-Laryngological Society of Alberta

President: Dr. R. F. Nicholls, McLeod Bldg., Edmonton.
Secretary: Dr. Mark R. Levey, Wells Pavillion, University Hospital, Edmonton.

Ophthalmological Society of Australia

President: Dr. Arthur H. Joyce, 55 Collins St., Melbourne.
Secretary: Dr. Arnold L. Lance, 135 Macquarie St., Sydney.
Place: Melbourne. Time: October 1949.

Ophthalmological Society of Egypt

President: Dr. F. Maxwell Lyons, Memorial Ophthalmic Laboratory, Giza.
Secretary: Dr. Mahmound Lutfi, Ophthalmic Hospital, Giza.
Place: Dar El Hekmah, 42, Kasr El Ainy, Cairo.

Ophthalmological Society of Hospital de Nuestra Señora de la Luz

Chairman: Dr. Manuel J. Icaza y Dublán, Artes 72, México, D. F., Mexico.
Secretary: Dr. Jorge Meyrán, Ezequiel Montes 135, México, D. F., Mexico.
Place: Hospital de Nuestras Señora de la Luz. Time: Second Friday of each month.

Ophthalmological Society of New Zealand

President: Mr. W. A. Fairclough, Imperial Bldgs., Queen St., Auckland.
Secretary: Dr. W. J. Hope-Robertson, Kelvin Chambers, 16 The Terrace, Wellington.

Ophthalmological Society of South Africa

President: Dr. M. Fine, 82 Lister Bldg., Jeppe St., Johannesburg.
Secretary: Dr. R. L. H. Townsend, National Mutual Bldg., Church Sq., Cape Town.

Ophthalmological Society of the United Kingdom

President: F. A. Juler, C.V.O., F.R.C.S.
Secretaries: Geoffrey J. O. Bridgeman, M.C., F.R.C.S. Dr. A. C. Cross, F.R.C.S.

Oxford Ophthalmological Congress

Master: Mr. F. A. Williamson-Noble, 27 Harley St., London W. 1, England.
Secretary-Treasurer: Dr. Ian C. Fraser, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford, England. Time: July.

Philippine Ophthalmological and Otolaryngological Society

President: Dr. Edmundo Reyes, 288 Gral. Solano, San Miguel, Manila.
Secretary-Treasurer: Dr. Carlos V. Yambao, Philippine General Hospital, Manila.

Polish Ophthalmological Society

President: Prof. Dr. W. Kapuściński, Libelta 14, Poznań.
Secretary: Dr. S. Topolski, Piusa 38, Warsaw.
Place: Ophthalmic Clinic, Oczki 6, Warsaw. Time: Every two years—Summer.

Puerto Rico Medical Association, Section of Ophthalmology and Otolaryngology

President: Dr. A. Navas Torres, Santurce.
Secretary: Dr. A. Laugier, San Juan.
Place: Asociacion Medica de Puerto Rico, Santurce. Time: Monthly.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Montague L. Hine, F.R.C.S.
Secretaries: Dr. Arthur Lister. Dr. H. E. Hobbs, 1 Wimpole Street, London, W.1.

São Paulo Society of Ophthalmology

President: Dr. Durval Prado, Rua Senador Paulo Egydio, 15-5°-514, São Paulo, Brazil.
Secretary: Dr. Rubens Belfort Mattos, Rua B. Stapetininga, 29 F-3° andar, São Paulo, Brazil.

Scottish Ophthalmological Club

President: Dr. Alexander Garrow, 15 Woodside Pl., Glasgow, C. 3.
Secretary: Dr. John Marshall, 11 Claremont Gardens, Glasgow, C. 3.
Place: Edinburgh and Glasgow, in rotation. Time: March and October.

Sociedad Argentina de Oftalmologia

President: Dr. Diego M. Arguello, Calle 46 No. 655, La Plata.
Secretary: Dr. Pedro F. Garcia Nocito, Vicente Lopez 1756 A, Buenos Aires.
Place: Buenos Aires. Time: Third Wednesday of every month.

Sociedade Brasileira de Oftalmologia

President: Dr. Natalicio de Farias, R. 13 de maio 23, 17° andar, Rio de Janeiro, Brazil.
Secretary: Dr. Ismar Pereira, Praca Floriano 55, 5° andar, Rio de Janeiro, Brazil.
Place: Third Friday every month from April to December.

Sociedad Colombiana de Oftalmologia y de Otorrinolaringologia

President: Dr. Jorge Suárez-Hoyos, Carrera 5a, no. 13-39, Bogotá, Colombia
Secretary: Dr. Fancisco Arango, Calle 20, no. 4-51, Bogotá, Colombia.
Place: Club Médico. Time: Second Tuesday of every month.
All correspondence should be addressed to the President.

Sociedad Chilena de Oftalmologia

President: Dr. Italo Martini Zerega, Pedregal, no. 216, Santiago, Chile.
Secretary: Dr. Adrian Araya Costa, Renaca, no. 34, Santiago, Chile.
Place: Santiago, Chile.

Sociedad Cubana de Oftalmologia

President: Prof. Lorenzo Comas, Calle D #461, Vedado, Habana, Cuba.
Secretary: Dr. Oscar F. Horstmann, Calle 5ta. #702, Vedado, Habana, Cuba.
Time: Second Tuesday of every month.

Sociedad Mexicana de Oftalmologia

President: Dr. Feliciano Palomino Dena, Londres 44, México, D. F.
Secretary: Dr. Teodulo Agundis Jr., Lucerna 63, México, D. F.
Place: Escuela Nacional de Medicina, Venezuela 4, México, D. F. Time: 8:30 p. m., first Tuesday of each month.

Sociedad Oftalmologia del Litoral, Rosario (Argentina)

President: Dr. Juan Manuel Vila Ortiz, Italia 663, Rosario.
Secretary: Dr. Maximo Carlos Soto, Rosario.
Place: Rosario. Time: Last Saturday of every month, April to November. All correspondence should be addressed to the President.

Sociedad de Oftalmologia de Córdoba

Chairman: Dr. Roberto Obregón Oliva.
Secretary: Dr. Alberto Urrets Zavalia (hijo), 27 de Abril 255, Córdoba, Argentina.

Sociedad de Oftalmologia de Guadalajara

President: Dr. Enrique Avalos Gonzales, Morelos 479, Guadalajara, Mexico.
Secretary: Dr. Jesus Gomez Plasencia, Pedro Moreno 587, Guadalajara, Mexico.
Place: Edifico Lutecia, Desp. 101. Time: Second Thursday of each month.

Sociedad de Oftalmologia del Litoral

President: Dr. Juan Manuel Vila Ortiz, Córdoba 1915, Rosario, Argentina.
Secretary: Dr. Carlos M. Soto.
Place: Rosario, Argentina. Time: Last Sunday in every month.

SOCIEDADE DE OFTALMOLOGÍA DEL NORTE

President: Dr. Jorge Luis Castillo, Mendoza 421, Tucumán, Argentina.
Secretary: Dr. Felix Berman, San Lorenzo 345, Tucumán, Argentina.
Place: Mendoza 421, Tucumán, Argentina.

SOCIEDADE DE OFTALMOLOGIA DE MINAS GERAIS

President: Prof. Hilton Rocha.
Secretary: Dr. Oswaldo Carvalho.
Place: Belo Horizonte, Minas Gerais, Brazil.

SOCIEDADE DE OFTALMOLOGIA E OTORINOLARINGOLOGIA DO RIO GRANDE DO SUL

President: Dr. Luiz Assumpção Osorio, Edificio Vera Cruz, Apartamento 134 Porto Alegre, Rio Grande do Sul.
Secretary: Dr. Fernando Voges Alves, Caixa Postal 928, Porto Alegre, Rio Grande do Sul.

SOCIEDADE DE OFTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DO BAHIA

President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

SOCIETA OFTALMOLOGICA ITALIANA

President: Prof. Giuseppe Ovio, Via Ombrone, 14 Roma.
Secretary: Prof. E. Leonardi, Piazza degli Eroi, 11 Roma.
Place: Roma Piazza degli Eroi 11.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. Edward Hartmann, Hospital Lariboisière, Paris 10.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. S. Larsson, Lund, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4, Stockholm.

SOUTHERN OPHTHALMOLOGICAL SOCIETY, ENGLAND

President: Dr. C. C. Schurr, 29 The Drive, Hove, Sussex.
Secretary: Mr. Nigel Cridland, 25 Craneswater Pk., Southsea, England.
Time: From March to October.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. A. Ray Irvine, Beverly Hills, Calif.
Secretary: Dr. Trygve Gundersen, 101 Bay State Rd., Boston.
Place: San Francisco. Time: June 26-30, 1950.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. C. H. McCaskey, 20 N. Meridian St., Indianapolis, Ind.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st. Ave. Bldg., Rochester, Minn.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Bernard Samuels, 57 W. 57th St., New York.
Secretary-Treasurer: Dr. Maynard C. Wheeler, 30 W. 59th St., New York 19.

Association for Research in Ophthalmology, Inc.

Chairman: Dr. Robert J. Masters, 23 E. Ohio St., Indianapolis.
Secretary-Treasurer: Dr. James H. Allen, University Hospitals, Iowa City.

Canadian Medical Association, Section on Ophthalmology

President: Dr. R. G. C. Kelly, 14 Lynwood Ave., Toronto.
Secretary: Dr. J. Clement McCulloch, 380 Medical Arts Bldg., Toronto.

Canadian Ophthalmological Society

President: Dr. Jules Brault, 418 Sherbrooke St. East, Montreal, Quebec.
Secretary-Treasurer: Dr. J. F. A. Johnston, 174 St. George St., Toronto 5.

National Society for the Prevention of Blindness, Inc.

President: Mr. Mason H. Bigelow, 1790 Broadway, New York 19.
Secretary: Dr. Franklin M. Foote, 1790 Broadway, New York 19.

SECTIONAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. Anthony M. Sellitto, 115 Connett Pl., South Orange.
Secretary: Dr. W. F. Grant, 1224 Salem Avenue, Hillside, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Ark-La-Tex Oto-Ophthalmic Society

President: Dr. W. Griffin Jones, 4421 Creswell, Shreveport, La.
Secretary: Dr. F. L. Bryant, Shreveport, La.
Place: Shreveport Club. Time: 6:30 p.m. first Monday, Sept. to June (incl.).

Central Illinois Society of Ophthalmology and Otolaryngology

President: Dr. Clifton S. Turner, 216 Parkside Dr., Peoria.
Secretary-Treasurer: Dr. Philip R. McGrath, Jefferson Bldg., Peoria.
Place: Various Central Illinois towns. Time: Two meetings a year.

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. Edward J. Zeiss, 103 W. College Ave., Appleton, Wis.
Secretary: Dr. G. L. McCormick, 650 S. Central Ave., Marshfield.

Hawaii Eye, Ear, Nose and Throat Society

President: Dr. Robert T. Wong, 1147 Union Street, Honolulu.
Secretary-Treasurer: Dr. Ogden Pinkerton, Young Hotel Bldg., Honolulu.
Place: Pacific Club, Honolulu. Time: Third Thursday of each month.

Inter-Mountain Oto-Ophthalmological Society

President: Dr. Ralph O. Porter, 50 North 1st East, Logan.
Secretary-Treasurer: Dr. Harry O. Frazier, 707 Medical Arts Bldg., Salt Lake.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month, September through May.

New England Ophthalmological Society

President: Dr. Benjamin Sachs, 520 Beacon Street, Boston.
Secretary-Treasurer: Dr. Garrett L. Sullivan, 101 Bay State Rd., Boston 15.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Wednesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Guy Boyden, 1735 N. Wheeler Ave., Portland 12, Ore.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco 8.
Place: San Francisco. Time: Spring, 1950.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Robert Wightman, 1114 Boylston Ave., Seattle, Wash.
Secretary-Treasurer: Dr. Barton E. Peden, 301 Stimson Bldg., Seattle 1, Wash.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Robert C. Fringer, Rockford News Tower, Rockford, Ill.
Secretary-Treasurer: Dr. Vernon C. Voltz, 806 Gas-Electric Bldg., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY & OTOLARYNGOLOGY

President: Dr. V. E. Cortopassi, 324 S. Washington Avenue, Saginaw, Mich.
Secretary: Dr. A. J. Cortopassi, 324 S. Washington Ave., Saginaw, Mich.
Place: Saginaw, Bay City or Flint, Mich. Time: Second Tuesday evening.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. M. W. Eggers, Sioux Falls, S. D.
Secretary-Treasurer: Dr. W. P. Davey, 2101 Grandview Blvd., Sioux City 18, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Murdock Equen, Atlanta, Georgia.
Secretary: Dr. Edley H. Jones, 1301 Washington Street, Vicksburg, Miss.
Place: Cincinnati, Ohio. Time: November 15-17.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. H. L. Brehmer, 221 W. Central Ave., Albuquerque, N. Mex.
Secretary: Dr. A. E. Cruthirds, 1011 Professional Bldg., Phoenix, Ariz.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. W. M. Dodge, 716 First National Bank Bldg., Battle Creek.
Secretary-Treasurer: D. Kenneth Lowe, 25 W. Michigan Ave., Battle Creek.
Time: Last Thursday of September, October, November, March, April and May.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Harold M. Griffith, Johnstown, Pa.
Secretary-Treasurer: Dr. Fred E. Murdock, 28½ W. Scribner St., Dubois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. C. G. Hinkle, Batesville, Arkansas.
Secretary: Dr. K. W. Cosgrove, 115 E. Capitol, Little Rock.
Place: Little Rock, Arkansas. Time: Second week in April.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. Leonard Swigert, Republic Bldg., Denver 2.
Secretary: Dr. James C. Strong Jr., 209 16th Street, Denver 2.
Place: University of Colorado Medical Center, Denver. Time: 3:00 p. m., third Saturday of each month, September to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. P. W. Snelling, 85 Jefferson Street, Hartford, Conn.
Secretary-Treasurer: Dr. Max Alpert, 881 Lafayette St., Bridgeport, Conn.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Lester A. Brown, 137 Doctors Bldg., Atlanta, Georgia
Secretary-Treasurer: Dr. B. E. Collins, 701 Elizabeth St., Waycross.
Place: Oglethorpe Hotel. Time: March 1950.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Byron Lingemann, Crawfordsville, Ind.
Secretary: Dr. Myron S. Harding, 23 E. Ohio St., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. C. A. Noe, 102-3rd Ave. S.E., Cedar Rapids, Iowa.

KANSAS STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Clifford Mullen, 1409 Minnesota Ave., Kansas City, Kans.
Secretary: Dr. Morris J. Ryan, 905 N. 7th St., Kansas City.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. William Wagner, 921 Canal Street, New Orleans, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

MEDICAL SOCIETY OF THE STATE OF PENNSYLVANIA, SECTION ON EYE, EAR, NOSE AND THROAT DISEASES

Chairman: Dr. William T. Hunt Jr., 1205 Spruce St., Philadelphia 7.
Secretary: Dr. Gabriel Tucker, 250 S. 18th St., Philadelphia 3.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Ralph H. Gilbert, 110 Fulton St. E., Grand Rapids.
Secretary: Dr. Walter Z. Rundles, 620 Maxine Ave., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Avery Prangen, Mayo Clinic, Rochester.
Secretary-Treasurer: Dr. Frank Adair, 822 Lawry Medical Arts Bldg., St. Paul 2, Minn.
Place: Minneapolis Club. Time: 6:00 p. m., second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. J. H. Herring, Lewistown.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

NEBRASKA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N St., Lincoln.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. A. M. K. Maldeis, 117 N. 6th St., Camden.
Secretary: Dr. Albert F. Moriconi, 438 Hamilton Ave., Trenton, N. J.
Place: Atlantic City. Time: May 1950.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Thomas H. Johnson, 30 W. 59th St., New York.
Secretary: Dr. Darrell G. Voorhees, 135 E. 65th St., New York 21.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. A. Harrill, Bowman Gray School of Medicine, Winston-Salem.
Secretary: Dr. MacLean B. Leath,

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. George C. Foster, Fargo.
Secretary-Treasurer: Dr. M. T. Lampert, Minot.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. C. W. Kuhn, 1020 S. W. Taylor St., Portland 5.
Secretary: Dr. Richard Fixott, 1020 S. W. Taylor St., Portland 5.
Place: Heathman Hotel, Portland. Time: 6:30 p. m., third Tuesday of each month.

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. D. S. DeSito, 1006 Highland Bldg., Pittsburgh, Pa.
Secretary: Dr. Benjamin F. Souders, 143 N. 6th St., Reading.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society, Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Pierre C. Jenkins, 155 Wentworth, Charleston.
Secretary-Treasurer: Dr. Roderick Macdonald, 330 E. Main St., Rock Hill.
Place: Greenville, South Carolina. Time: Sept. 12-15.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George Burchfield, Maryville.
Secretary-Treasurer: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. August J. Streit, 811 Fisk Bldg., Amarillo, Texas.
Secretary: Dr. John L. Matthews, 414 Navarro Street, San Antonio.
Place: Gunter Hotel, San Antonio. Time: Dec. 23, 1949.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Francis H. McGovern, 105 S. Union St., Danville.
Secretary: Dr. Peter Pastore, Richmond, Va.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Garnett P. Morison, Charles Town, W. Va.
Secretary: Dr. Melvin W. McGehee, Huntington, W. Va.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. V. C. Malloy, Second National Bldg., Akron 8, Ohio.
Secretary-Treasurer: Dr. A. L. Peter, 406 Akron Savings & Loan Bldg., Akron 8, Ohio.
Place: Akron City Club. Time: 6:30 p. m., first Monday in January, March, May and November.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Russell Burke, 490 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. James T. King, 384 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine. Time: 7:30 p. m., fourth Monday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

President: Dr. James I. Moore, 11 E. Chase St., Baltimore 2.
Secretary: Dr. Elliott Randolph, 11 E. Chase St., Baltimore 2.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. David A. McCoy, Woodward Bldg., Birmingham, Ala.
Place: Thomas Jefferson Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. Frank E. Mallon, 1 Hanson Pl., Brooklyn 16.
Secretary-Treasurer: Dr. Louis Freimark, 256 Rochester Ave., Brooklyn 13.
Place: Brooklyn Eye and Ear Hospital, Greene Ave. and Cumberland St. Time: 8:15 p. m., third Thursday in February, April, October and December.

Buffalo Ophthalmologic Club

President: Dr. Arthur L. Bennett, 147 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Herbert R. Reitz, 446 Linwood Ave., Buffalo 9.
Place: Park Lane. Time: Second Thursday of each month from October to May.

Central New York Eye, Ear, Nose and Throat Society

President: Dr. Everett Wood, Auburn, N. Y.
Secretary-Treasurer: Dr. Alfred W. Doust, 306 State Tower Bldg., Syracuse.
Place: Syracuse. Time: Quarterly.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. Willard H. Steele Jr., 552 McCallie Ave., Chattanooga, Tenn.
Place: Mountain City Club. Time: Third Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Derrick Vail, 700 N. Michigan Ave., Chicago.
Secretary: Dr. J. R. Fitzgerald, 3215 W. North Avenue, Chicago.
Place: Illini Union Bldg. Time: 7:30 p. m., third Monday of each month from October to May.

Cincinnati Ophthalmologic Club

Chairman: Rotate alphabetically.
Secretary: Dr. Josef D. Weintraub, Provident Bank Bldg., Cincinnati 2, Ohio.
Place: As announced. Time: 8:00 p. m., second Friday of each month from November to May, inclusive.

Cleveland Ophthalmological Club

President: Dr. G. L. Miller, 14805 Detroit Avenue, Lakewood, Ohio.
Secretary: Dr. W. P. Chamberlain, 7405 Detroit Avenue, Cleveland, Ohio.
Place: Hotel Statler, Cleveland. Time: Second Tuesday in November, January, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

President: Dr. Perce de Long, 37 S. 20th St., Philadelphia.
Clerk: Dr. M. Luther Kauffman, Medical Arts Bldg., Jenkintown, Pa.
Place: College of Physicians Bldg. Time: 8:15 p. m., third Thursday of every month from October to May, inclusive.

Columbus Eye, Ear, Nose and Throat Society

President: Dr. Claud Perry, 40 South 3rd Street, Columbus, Ohio.
Secretary-Treasurer: Dr. Vernon D. Stephens, 9 Butler Avenue, Columbus, Ohio.
Place: University Club. Time: 6:00 p. m., first Monday of each month, from October to May, inclusive.

Corpus Christi Eye, Ear, Nose and Throat Society

President: Dr. B. B. Friedman, Jones Bldg., Corpus Christi, Texas.
Secretary: Dr. S. K. Stroud, Medical Professional Bldg., Corpus Christi, Texas.
Place: Nueces Hotel. Time: 6:30 p. m., third Tuesday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. A. Hoefer, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. W. B. Wilkinson, Tenth and Zangs, Dallas, Texas.
Place: Melrose Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. Byron Merkel, 1112 Equitable Bldg., Des Moines, Iowa.
Secretary-Treasurer: Dr. H. H. Gurau, 213 Bankers Trust Bldg., Des Moines, Iowa.
Place: Des Moines Club. Time: Second Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Wesley G. Reid, 974 Fisher Bldg., Detroit 2.
Place: Wayne County Medical Society. Time: Second Monday of each month, November to April, inclusive.

Detroit Ophthalmological Society

President: Dr. Aaron Ricker, Pontiac, Mich.
Secretary: Dr. A. P. Wilkinson, 974 Fisher Bldg., Detroit 2.
Place: L'Aiglon-Fisher Bldg. Time: 6:30 p. m., third Thursday of each month from November to April, inclusive.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. John H. Sulzman, 1831 Fifth Ave., Troy, N. Y.
Secretary-Treasurer: Dr. E. Martin Freund, 762 Madison Ave., Albany 3.
Place: Albany, Troy, Schenectady, rotating monthly. Time: 8 p. m., first Thursday of each month, from October to June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. John Eschenbrenner, 306 W. Broadway, Fort Worth, Texas.
Secretary-Treasurer: Dr. C. Keith Barnes, 921 Neil P. Anderson Bldg., Fort Worth 2, Texas.
Place: All Saints Hospital. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Ophthalmological and Oto-Laryngological Section

President: Dr. Norma B. Elles, Niels Esperson Bldg., Houston, Texas.
Secretary: Dr. R. Marion Johnson, Medical Arts Bldg., Houston, Texas.
Place: River Oaks Country Club. Time: 6:30 p. m., second Thursday of each month from October to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. Kenneth L. Craft, 1002 Hume Mansur Bldg., Indianapolis 4.
Secretary: Dr. J. Lawrence Sims, 809 Hume Mansur Bldg., Indianapolis 4.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. John McLeod, Argyle Bldg., Kansas City, Mo.
Secretary: Dr. James W. May, 906 Grand Ave., Kansas City, Mo.
Time: 6:00 p. m., third Thursday of each month from November to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Orville W. Cole, 125 E. 8th St., Long Beach 2, Calif.
Secretary: Dr. Edmund Godwin, 117 E. 8th St., Long Beach, Calif.
Place: Seaside Memorial Hospital. Time: 6:15 p. m., third Wednesday of each month from October to May.

Los Angeles Ophthalmological Society

President: Dr. William Endres, 523 W. 6th St., Los Angeles, Calif.
Secretary: Dr. Robert A. Norene, 727 W. 7th St., Los Angeles 14.
Place: 3550 Wilshire Blvd., Los Angeles. Time: 6:30 p. m., third Tuesday of each month, September through June.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 5.
Secretary-Treasurer: Dr. Victor Goodhill, 2007 Wilshire Blvd., Los Angeles 5.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye and Ear Society

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Lower Anthracite Eye, Ear, Nose and Throat Society

Chairman: Each member in alphabetical order.
Secretary: Dr. James J. Monohan, 31 S. Jardin St., Shenandoah, Pa.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.
Secretary: Dr. Frazier Williams, 1801 I St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

Milwaukee Oto-Ophthalmic Society

President: Dr. S. S. Blankstein, 208 E. Wisconsin Avenue, Milwaukee 2.
Secretary-Treasurer: Dr. J. P. Wild, 161 W. Wisconsin Avenue, Milwaukee.
Place: Athletic Club. Time: 6:30 p. m., fourth Tuesday of each month from October to May.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel: Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. B. Alexander, Medical Arts Bldg., Montreal 25, Canada.
Secretary: Dr. R. Cloutier, Medical Arts Bldg., Montreal 25, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

President: Dr. James B. Miller, S. Garden Street, Columbia, Tenn.
Secretary: Dr. N. B. Morris, 647 Doctors Bldg., Nashville 3, Tenn.
Place: James Robertson Hotel. Time: 6:30 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President and Secretary: Dr. Mercer G. Lynch, Ochsner Clinic, New Orleans 15, La.
Place: Charity Hospital. Time: 8 p. m., first Tuesday of every month.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Wendell L. Hughes, 131 Fulton Ave., Hempstead, N. Y.
Secretary: Dr. Isadore Givner, 108 E. 66th St., New York 21.
Place: Academy of Medicine. Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Sidney A. Fox, 63 E. 75th Street, New York.
Secretary: Dr. Leon Ehrlich, 211 Central Park W., New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Oklahoma City Academy of Ophthalmology and Otolaryngology

President: Dr. Charles A. Royer, 1221 N. W. 19th St., Oklahoma City.
Secretary: Dr. C. W. McClure, 1005 Medical Arts Bldg., Oklahoma City.
Place: University Hospital. Time: 7:30 p. m., first Tuesday of each month from September to June.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. T. T. Smith, 211 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. George T. Alliband, 1020 Medical Arts Bldg., Omaha 2.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m. dinner; 7 p. m. program; third Wednesday of each month from October through April.

Passaic-Bergen Ophthalmological Club

President: Dr. Thomas Sanfacon, 340 Park Ave., Paterson, N. J.
Secretary-Treasurer: Dr. J. Averbach, 435 Clifton Ave., Clifton, N. J.
Place: Doctors Lounge, Paulsen Medical and Dental Bldg. Time: 8 p. m., last Tuesday of each month except June, July and August.

Philadelphia County Medical Society, Eye Section

President: Dr. Alfred Cowan, 1930 Chestnut Street, Philadelphia 3, Pa.
Secretary: Dr. L. Waller Deichler, 1930 Chestnut St., Philadelphia 3.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. John S. Plumer, 121 University Place, Pittsburgh.
Secretary: Dr. Samuel D. Evans, 1501-1502 Park Bldg., Pittsburgh 22.
Place: Pittsburgh Academy of Medicine Bldg. Time: 8:00 p. m., fourth Monday of each month, except June, July, August and September.

Reading Eye, Ear, Nose and Throat Society

President: Dr. W. J. Hertz, 125 N. 8th Street, Allentown, Pa.
Secretary: Dr. Paul C. Craig, 232 N. 5th St., Reading, Pa.
Place: Wyomissing Club. Time: 6:00 p. m., third Wednesday of each month from September to July.

Richmond Eye, Ear, Nose and Throat Society

President: Dr. Clifford A. Folkes, Professional Bldg., Richmond, Va.
Secretary: Dr. W. F. Bryce, 4920 New Kent Road, Richmond, Va.
Place: Commonwealth Club. Time: 6 p. m., firth Tuesday of January, March, May and October.

Rochester Eye, Ear, Nose and Throat Club

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.

St. Louis County Medical Society, Section of Eye, Ear, Nose and Throat

President: Dr. Orien B. Patch, 205 West 2nd St., Duluth, Minn.
Secretary: Dr. J. C. O'Neill, 205 West 2nd St., Duluth, Minn.
Place: St. Mary's Hospital, Duluth. Time: Second Thursday of each month from September through May.

St. Louis Ophthalmic Society

President: Dr. Daniel Bisno, 4500 Olive Street, St. Louis 8.
Secretary: Dr. J. H. Bryan, 3720 Washington Blvd., St. Louis 8.
Place: Elliott Auditorium, McMillan Hospital. Time: Fourth Friday of each month from October to April, inclusive, except December, at 8:00 p. m.

San Antonio Society of Ophthalmology and Otolaryngology

President: Dr. A. A. Nesbit, South Texas Bank Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. Fred B. Cooper, 827 Medical Arts Bldg., San Antonio 5, Texas.
Place: San Antonio, Texas, Brooke General Hospital and Randolph Field. Time: 7 p. m., second Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. William Duggan, 490 Post St., San Francisco 2.
Secretary: Dr. George Campion, 490 Post St., San Francisco 2.
Place: Society's Bldg., 2180 Washington St., San Francisco 9. Time: 8:15 p. m., fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. Ralph Riggs, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. Frank L. Bryant, 2622 Greenwood Rd., Shreveport 2, La.
Place: Shreveport Club. Time: 6:30 p. m., first Monday of every month except June, July and August.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Phillip B. Greene, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. Cornelius E. Hagan Jr., Old National Bank Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Bldg. Time: 8 p. m., fourth Tuesday of each month except, June, July and August.

Toledo Eye, Ear, Nose and Throat Society

President: Dr. James Cooper, 316 Michigan Street, Toledo 2, Ohio.
Secretary: Dr. William H. Bonser, 219 15th Street, Toledo, Ohio.
Place: Toledo Club. Time: 8:00 p. m., second Tuesday of each month.

Toronto Academy of Medicine, Section of Ophthalmology

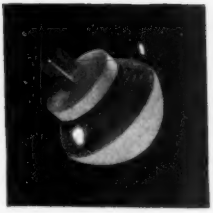
Chairman: Dr. A. L. Morgan, Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. J. C. Hill, Medical Arts Bldg., Toronto 5, Canada.
Place: Academy of Medicine, 288 Bloor St. W. Time: 8:00 p. m., second Tuesday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. Jerome A. Sansoucy, 2017 Massachusetts Ave. N. W., Washington, D. C.
Secretary: Dr. Thomas A. Egan, 1835 Eye St. N. W., Washington, D. C.
Place: Hotel Kennedy-Warren. Time: 6:00 p. m., first Monday of November, January, March and May.

Wilkes-Barre Ophthalmological Society

President: Dr. Joseph V. Connole, 72 W. Union Street, Wilkes-Barre, Pa.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Luzerne County Medical Society. Time: 8:30 p. m., last Tuesday of each month from October to May.



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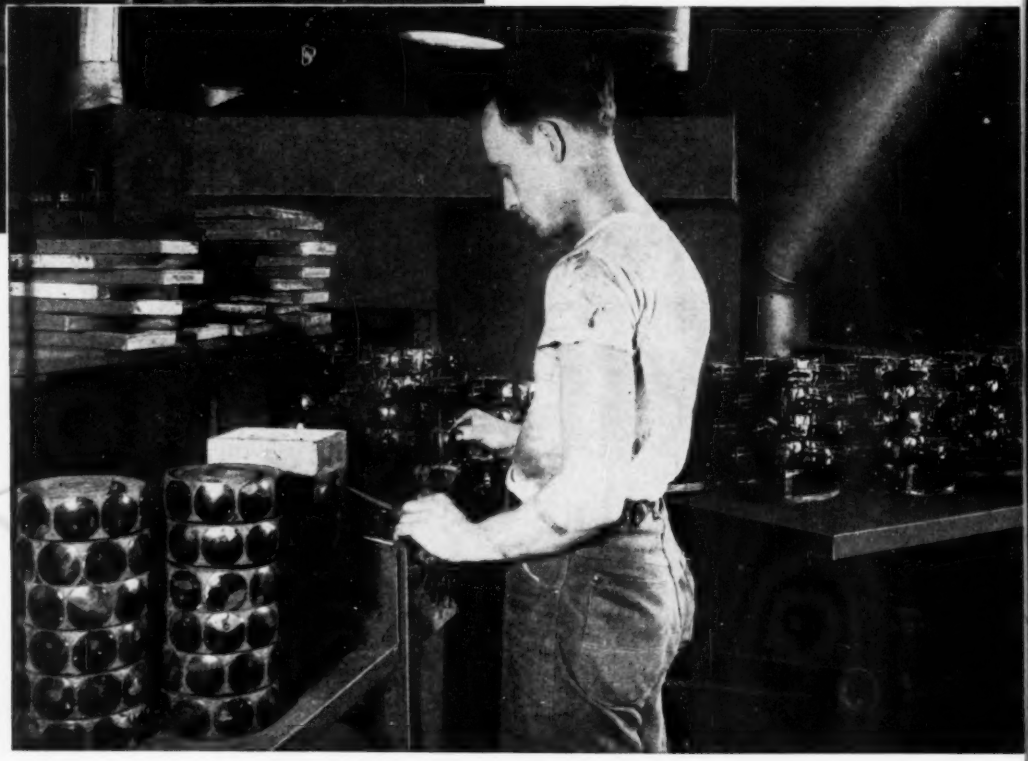

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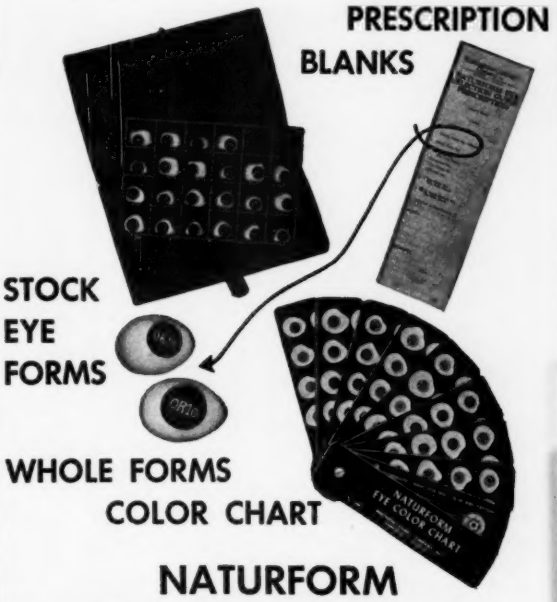

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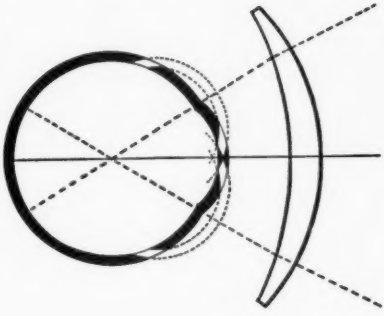

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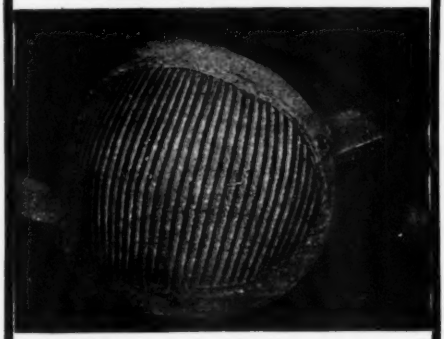

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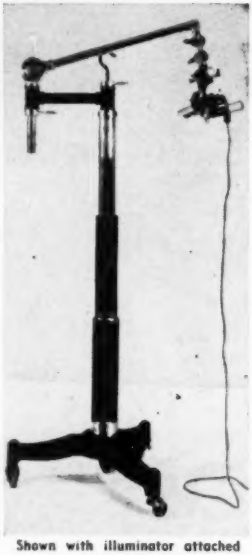

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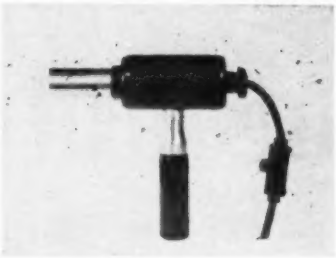

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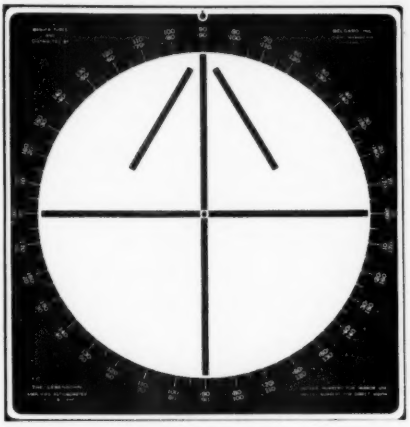

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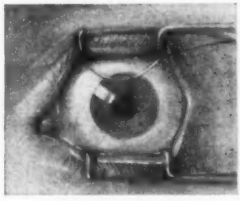

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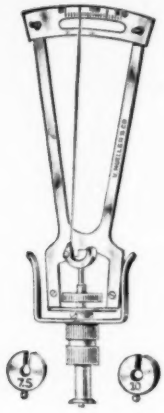

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